AN OPEN CLINICAL STUDY ON

KALLADAIPPU (UROLITHIASIS)

WITH THE EVALUATION OF SIDDHA DRUG

VEDIYUPPU CHENDOORAM

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CERTIFICATE

This is to certify that the dissertation entitled "**AN OPEN CLINICAL STUDY ON KALLADAIPPU"** is a bonafide work done by **Dr.P.GOVINDAMMAL**, Government Siddha Medical College, Chennai – 600 106 in partial fulfilment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2013 – 2016.

Name & Signature of the Guide

Name & Signature of the Head of Department

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CONTENTS

INTRODUCTION

INTRODUCTION

"வாழ்த்த வல்லார் மனத்துள்உறு சோதியை
தீர்த்தனை அங்கே திளைக்கின்ற தேவனை
ஏத்தியும் எம்பெருமான் என்று இறைஞ்சியும்
ஆத்தம் செய்து ஈசனை அருள் பெறலாமே"[1]

Siddha system is a traditional medical system of south India. The art of healing incorporates a variety of holistic practices and remedies which were established by the Siddhars, out of their intuitions. Hence it is named as Siddha Medicine.

According to siddha theory the human body is made by 96 thathuvam on the principle of panjapootham and thridhosam. Siddhars used thiridhosam- panjapootham-arusuvai relativity to cure the disease.siddhars used herbs,metals minerals as their ingredients in their prepartion[2].

Yugi munivar spread this knowledge to human beings for the welfare of their wellbeing.This has been quoted by yugi munivar by the following lines,

சொல்லிடவே தேவிக்குச் சதாசிவன்றான்
சொல்லவே தேவியும் நந்திக்குச் சொல்ல
நயமுடன் தன்வந்திரி அசுவினிக்குச் சொல்ல
அல்லிடவே அசுவினியாந் தேவர் தாமும்
அகத்தியருக் குரைத்திட வேயம் முனீந்திரன்"[3]

As per siddha aspect, the physiological function of the human body is maintained by three thodams called as vatham, pitham and kabam. The normal proportion of naadi is 1:1/2:1/4 respectively, any change in this proportion leads to many diseases.

Siddhars classified 4,448 types of disease. Within that kalladaippu is one of the disease commonly affecting men than women. (4)

Even though many siddhars explain about diseases, **Yug**i in his **"YUGI VAIDHYA CHINTHAMANI 800"** elaborately said the aetiology, pathology, classification, clinical features, and prognosis of kalladaippu. The diseases of urinary system are divided into two types, They are,

"நீரினை அருகல் நோய்

நீரினை பெருகல் நோய்"

The kalladaippu comes under the classification of "நீரினை அருகல் நோய்" which produces low urine output and dries up the urine and form urinary calculi , due to various aetiological factors.[5]

Nephrolithiasis or urolithiasis is formation of urinary calculi at any level of the urinary tract. Urinary calculi are worldwide in distribution but are particularly common in some geographic locations such as in parts of the United States, South Africa, India and South- East Asia. It is estimated that approximately 2% of the population experiences renal stone disease at some time in their life with male- female ratio of 2:1. The peak incidence is observed in 2nd to 3rd decades of life. Renal calculi are characterized clinically by colicky pain.[6]

நோய்நாடி நோய்முதல் நாடி அதுதணிக்கும்

வாய்நாடி வாய்ப்பச் செயல்"[7]

As per Thiruvalluvar says, a good physician must find out the disease with its root cause and cure it with the help of his diagnostic tools.

In our system, there are many drugs indicated for kalladaippu disease. THIRUMOOLAR Says about the siddha medicines as follows:

"வீர மருந்தென்றும் விண்ணோர் மருந்தென்றும்

நாரி மருந்தென்று நந்தியருள் செய்தான்

ஆதி மருந்தென் றறிவா ரகலிடஞ்

சோதி மருந்திது சொல்லவொண் ணாதே3

The **vediyuppu chendooram** has been selected for the clinical study to prove the efficacy of the medicine in kalladaippu. A proper dosage, anupanam and pathiyam were also followed to get good prognosis and to prove the efficacy of the trial drug in this disease.

AIM AND OBJECTIVES

AIM

To study the efficacy of the siddha medicine **veddiyuppu chendooram** both clinically and experimentally for **kalladaippu.**

OBJECTIVES

- To study the evaluation of siddha trial drug vediyuppu chendooram for kalladaippu
- To evaluate the safety profile of the trial drug.
- To collect the literature of the both siddha and modern aspect related to the disease.
- To study the disease with deep observation on aetiology, classification, pathology, diagnosis, differential diagnosis, prognosis and treatment by siddha aspect.
- To gather the diagnostic knowledge by mukutram, udal thathukkal, uyir thathukkal, and en vakai thervugal.
- To make a clinical observation about the disease in relation of age, sex, occupation, socio economic status.
- To use modern parameters to confirm the disease.
- To evaluate
    - Toxicological analysis-acute and subacute study
    - physico chemical analysis
    - pharmacological analysis –lithotriptic activity and
    - biostatisical analysis of the trial drug.
- The haematological analysis, urine analysis, ultra sonogram radiological studies will be done to all patients.
- All patients are subjected to thorough investigation before and after treatment.

REVIEW OF LITERATURE

SIDDHA ASPECT

REVIEW OF LITERATURE

SIDDHA ASPECT

Theran karisal says,

The diseases of the urinary system are divided into two types. They are

"நீரினை அருகல் நோய்

நீரினை பெருகல் நோய்"[5]

The disease kalladaippu comes under the classification of "Neerinai Arukkal Noi". In siddha system of medicine, the disease kalladaippu is mentioned by Yugi Munivar in Yugi Vaidhya Chinthamani 800.

"நீரிரு வினைக் குணத்தை நீயறி விரித்துச் சொல்வாம்

நீரினை பெருக்க லொன்றே நீரினை யருக்க லொன்று

நீரிழிவுடனே கொல்லும் நீர்க்கட்டு வினைகளொன்று"[9]

KALLADAIPPU NOI

VERUPEYAR (SYNONYMS)

Achmari

IYAL (DEFINITION)

Sudden obstruction in the flow of urine, pain at the tip of the penis in males and clitoris in females, burning micturition, loin to groin pain, passing of small sand like stones along with urine are the cardinal features of this disease. [10]

Dehydration occurs due to overheat of the body. It leads to solid or crystalline aggregation from the dietary minerals in the kidney. The formed stone cannot expelled by kidnys leads to this diseased condition.[11]

Large concretions of stone in the bladder or kidney are known as calculus or gravel. It is attended with difficulty in passing urine.[12] Sudden obstruction to the flow of urine, pain at the tip of the penis in males and clitoris in females, burning micturition, pain in loin to groin region.[13]

விலகு சிலநேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுமொது கினால் நோகாது
வழுகிய மந்த்தால் வாயுவந்தே புகில்
கழுவி முதிர்ந்திடும் கல்லடைப்பாகும்.[14]

தானான மூத்திரப்பை தன்னிலப்பா
தனியான கல்லிருந்தால் சொல்லக் கேளே
ஊனான மூத்திரந்தா னடிக்கடியே தானும்
உள்ளபடி இறங்குமடா சொன்னேன் பாரு
தேனான கல்லப்பா தாரை மேலே
தொப்பெனவே விழுவதால் தாரை யப்பா
மானாக விழுவதனாலே நின்று போகும்
மக்களுக்கு இதுதீர சலாகை போடே[15.]

“தானென்ற மூத்திரத்தால் நறநறவென்று
தங்கியதோர் பொடியேனும் மணல் தானப்பா
வானென்ற சிறியதொருகல்லா வதப்பா
வளமாக வந்து விழும் நோய்க்குத்தானே
ஏனென்று அச்மரீரோக மென்ற பேராம்
தாக கல்லுகள்தான் விழுகும்போது
கோனென்று குண்டிக்காய் மூத்திரக் குழலப்பா
குணமான மூத்திரப்பை நீர்த்தாரைக்கேளே

கேளடா முங்குறியில் எரிச்சல் கண்டு
கொடியாக வேதனைகள் காட்டுமப்பா
வாளடா சிறியதொரு கற்கள் தானே
வளமான மூத்திரப்பை குழல் வழிப் படியாய்த்
தேளடா வரும்போது திரே கந் தன்னில்
தெரிப்பது போல் யிரு வேதனை செய்யும் பாரு
நாளடா கற்கள் தானிறங்கி விட்டால்
நலமான வேதனைகள் தீரும்பாரே" [15]

If a stone present in the bladder, there will be frequency of Micturition.when the stone descends to urethra there will be obstruction of urine flow. It is cured through catheterization.

NOI VARUM VAZHI (AETIOLOGY)

"தெளிந்ததோர் கல்லடைப்புற் பத்தி கேளாய்
சிறிதுநாட் டுடங்கியே மேகந் தன்னால்
தளிந்ததோர் சலப்பையிலு திரந் தோய்ந்து
சந்து சந்தாகவே பருத்துக் கொள்ளும்
வளிந்ததோர் வாதபித்தங் கோபித் தக்கால்
வந்து பெருங்கல்லாய் நீர்வழி யடைத்து
"நளிந்ததோர் நாலுவிதக் கல்லடைப்பு
நண்பான வரலாறு நாட்டக் கேளே"

கலங்கினதோர் தண்ணீர்தான் குடித்த பேர்க்கும்
கல்லெலும்பு மயிர் மண்தான் கலந்தன் னத்தில்
அலங்கின தோரன்னங்க ளருந்த லாலும்
அழுகலோடு மூத்தபண்ட மருந்த லாலும்
மலங்கினதோர் மாப்பண்ட மருந்தலாலும்
மந்தத்தில் வாய்வானப தார்த்தந் தன்னை
துலங்கின தோருசிதன்னிற் சுவைத்தலாலும்
சுருக்காய் கல்லடைப்புவந் துதோன்றுந் தேனே."[16]

The urine constituents will easily deposit on the urinary tract and form the stone. At that time by vitiation of vatham and pitham these small stone becomes larger in size and block the urinary passage. The semen will stagnate for a long time in the urinary tract, so it will obstruct the urine flow Urinary stone are also formed due to the drinking of contaminated hard water, taking of food mixed with sand and small stones consuming of contaminated food articles, food containing more carbohydrates, unhealthy food habits.

A urinary disease occasionally developed in the urinary bladder which is known as vesical calculus. It is said to be due to the deranged vayu encircling or prevailing in the region of the abdomen arising from any of the following causes.

1) Suppression of seminal discharge during sexual intercourse.
2) Retention of semen in the spermatic region in involuntary discharge during nocturnal emissions due to excessive heat in the body.

The calculus are stone which is formed in the bladder may vary in size from that of the particles of sand or mustard upto things as large as green gram or Bengal gram and sometimes attains the size of a hen's egg even and block the passage of urine. It is accompanied by pain and difficulty in passing urine. [17]

"நீரினைத் தடுத்தல் செய்யின்
நீர்க்கட்டுத் துவாரம் புண்ணாம்
பாறிடு சந்து சந்தில்
பண்பற நோவதாகும்
நேரிலங் கயரும் காமியம்
நிச்சயம் நோதல் செய்யும்
பாரினிலபான வாயு
பண்புறச் சேருமன்றே"[18]

POTHU KURIGUNANGAL (Clinical features)

- Gradual or sudden obstruction of the urine flow.
- Unbearable pain in the penis
- Excruciating pain and swelling is experienced at the tip of penis if the calculus attempts to expel.
- Colicky pain radiating from loin to groin lower abdomen and urethra if the calculus is irregular with sharp projection.
- Burning and scanty micturition and Haematuria.[19]

CLASSIFICATION

Classification according to yugimamunivar,

"தோன்றினதார் நாலினிட நாமங் கேளாய்
சுறுக்கான வாதத்தின் கல்லடைப்பு
பூன்றியதோர் பித்தத்தின் கல்லடைப்பு
புரண்டதோர் சேத்துமத்தின் கல்லடைப்பு
தீன்றியதோர் தொந்தமாங் கல்லடைப்பு
தேகத்திற் பற்றியேசி றிதுகாலம்
தான்றியே சலப்பையில் வந்தி ழிந்து
சருவியே லிங்கத்திற்ற ரிக்குந் தானே" [20]

According to Yugi vaidhya chinthamani, kalladaippu is classified into four types.

1) Vadha kalladaippu
2) Piththa kalladaippu
3) Silethuma kalladaippu
4) Thondha kalladaippu

1) Vadha kalladaippu:

"தரித்து நாபிக்குங்கீழ் சுருக்காய் குற்றில்
சலமலந்தான் விழாமற் றம்ப மாகி
வரித்துமே லிங்கத்தில் வலியுமாகி
மருவியதோர் பொத்தியெலாஞ் சுரந்து கட்டி
திரித்தியே கிடைக்கொடாப் பிரட்டலாகித்
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்தோர் சதைபோல உவர்ப்பு மாகும்
ஓங்கியதோர் வாதக் கல்லடைப்பு தானே."[20]

Acute pricking pain in the lower abdomen, scanty Micturition, obstruction to the flow of urine, pain in the penis, abdominal discomfort, and albuminuria will be present with mucous discharge and black coloured stone will be expelled.

2)Piththa kalladaippu:

"அடைப்பாகிச் சலந்தானு மருவலாகி
அயங்காச்சி சொருகினாற் போலே காணும்
புடைப்பாகிப் பொற்றியெங் கும்பு ழுக்கமாகிப்
பூட்டுபோல் பிகுவாகிப் பிரட்டலாகும்
மடைப்பமாகி உதிரநிற மாய்க்கல் லாகி
வந்திழிந்து லிங்கத்தில் மாட்டிக் கொள்ளும்
குடைப்பாகிக் குற்றலாய்க் கூச்சலாகிக்
குதட்டுமே பித்தக்கல் லடைப்பு தானே." [21]

Obstruction of urine flow, pricking pain and burning sensation in external meatus, expulsion of blood coloured stones.

3)Silethuma kalladaippu:

"தானான தொப்புளிலே வில்லு போலச்
சலியாமற் சுரந்துமே சற்றே குற்றும்
ஏனான காலோடு கைகள் சந்து
இடுப்புதான் குடைசலாயி சிவு காணும்
வேனான லிங்கத்தின் வெண்மை தன்னில்
விறுவிறென் றேகடுப்பாகி வியற்வை யாகும்
தேனான வெளுப்புக்கல் சிறுகல் லாகச்
சிக்கலாய் வந்திறங்குச் சேட்பந் தானே"[21]

Pricking pain in umbilicus, pain in the joints of hands and legs, expulsion of white coloured stone in urine, excessive sweating.

5) Thondha kalladaippu:

"வந்திறங்கும் நீர்தாரை யடியிற் றானும்
மாவருத்த முண்டாகி வலியுமாகி
நொந்திறங்கி நீர்தானு மருவிபாயும்
நொய்தான சிறுமணல் போல் நொறுங்கிக் கல்லான்
சந்திறங்கி நீர் வழியில் வந்து விழும்
தாக்கான சிறங்கைக்கல் தினமொன்றுக்கு
துந்திறங்கித் தினந்தினமு மிழந்து கொல்லும்
தொந்தமாங் கல்லடைப்புச் சூட்டிட் டாயே"[22]

Severe pain in urethra, Dysuria, Oliguria, handful of small sand like stones will expel with severe pain.

According to Dhanvanthri,

"திருந்திய வாதபித்தச் சிலேற்பனம் பிரகோபித்தால்
வகுந்தக மரித்தா நான்கு வகைப்படும் கல்லரிப்பான்
பிரிந்திடுஞ் சிலேற்பனாக மரிபித்தா சுமரி பின்னு
மிருந்திடு சுக்கிலாசு மரி நான்கு மெய்து மென்றே"[23]

Achmari is classified into four types,

1) Kallerippan
2)Silethuma achmari
3)Piththa achmari
4)Sukkilachmari

1) **Kallerippan gunam:**

"சுத்துநீர் நாலிந்தன்னிற் சுக்கிலந் தனிற் சிலேற்பம்
பித்துமீது லர்த்தல் கல்லாய்ப் பீசகிநீ ரடைத்துக் கொள்ளுங்
கொத்து நீரிற்றுவிழுங் கொப்புளநோ குடம்பு காயுஞ்
சித்தமா யருசி யுண்டாஞ் சேர்ந்தகல் லெரிப்பனாமே."[23]

Formations of stones in the urinary tract, oliguria, pricking pain around the umbilicus, fever, anorexia are the symptoms of this type.

2) **Silethuma achmari gunam:**

நீர் வருநாளந் தன்னில் நின்றுநீர் சிறுத்துக் கொண்டு
சோர்தரும் சிவப்பு வெண்மை சுக்கிலம் போலவீழும்
பேர்பெற நாலா மெட்டுப் பின்னமாய்க் கல்லுவீழும்
ஏர்பெறு சிலேற்பனத்தில் அச்சமரி என்னலாமே" [24]

Oliguria due to obstruction of stone in urethral orifice, stones can be expelled out into pieces.

3) **Piththa achmari gunam**:

"பெய்யும் நீர் நாளந் தன்னில் பித்தத்தா லெரிப் பெழுந்து
செய்யுவுஷ் ணத்தால் வெந்து சேங்கொட்டை போற்கல் லுண்டாம்
நய்யவே தனைகள் செய்யும் நவில் குணம் பித்தந்தன்னில்
எய்தசு மரியென்றே முன்னியம் பினரறிவின் மிக்கோர்." [24]

Burning micturition in urethral orifice, formations of stones, severe pain are the symptoms

4)**Sukkilachmari gunam:**

"சுக்கிலம் வருங்காலத்தில் தம்பித்தாற் சுக்கிலந்தான்
மிக்ககல் லாகிவெம்பி விதனமாய் நீர்விடாமற்
சிக்கிநீர் விழாமலங்கே மணல் வீழும் வெளுக்குந்தேகம்
மிக்குணஞ் சுக்கிலாசு மரியசாத் தியமென்றோதே."[25]

Stagnation of semen leads to the formation of stones, oliguria, and expulsion of small sand like stones. It is fatal.

SAATHIYAM, ASAATHIYAM (PROGNOSIS)

As per yugi,

Vatha kalladaippu, piththa kalladaippu, silethuma kalladaippu are curable. Thondha kalladaippu is fatal.

"சூட்டிய சாத்தியத்தைச் சொல்லக் கேளாய்
சுளுக்காகும் வாதத்தின் கல்லடைப்பு
பூட்டிட்ட பித்தத்தின் கல்லடைப்பு
புகழான சேட்டுமத்தின் கல்லடைப்பு
மூட்டிட்ட இதுமூன்று மசாதியமாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்லடைப்புத்
தொடு சுறவே கொல்லுமிது சூட்சந்தானே."[26]

MUKKUTRA VAERUPADUGAL (PATHOGENESIS):

Disease occurs due to the derangement in

- Uyir thathukkal
- Udal thathukkal
- Kalamarupadu (seasonal changes)
- Thinai (living lands) and
- Udal vanmai.

Mukkutra iyal:

The function of the three uyir thathus:

a) Vali – Kattru + Veli
b) Azhal – Thee
c) Iyyam – Neer + Mann

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1:1/2:1/4) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

VATHAM

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations it is classified into ten types. They are tabulated below.

S.No	Vatham	General Features	Changes in Kalladaippu
1	Piranan (Uyirkkaal)	Responsible for respiration and it is necessary for proper digestion.	Normal
2	Abanan (Keel nokkukkaal)	Responsible for all the downward forces such as voiding of urine, stools, semen, menstrual flow.	Affected (scanty Micturition)
3	Viyanan (Paravukaal)	Dwells in the skin and is concerned with the sense of touch, extension and flexion of the parts of the body and distribution of the nutrients to various parts of the body.	Normal
4	Uthanan (Melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc.,	Affected (Nausea, vomiting)
5	Samanan (Nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ.	Affected
6	Nagan	Helps in opening and closing of eyelids.	Normal
7	Koorman	Responsible for vision, lacrimation and yawning.	Normal
8	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing.	Normal
9	Thevathaththan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc.,	Normal
10	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3 days of death, forming a way through the skull[27]	Normal

PITHAM

It is the thermal life force of the body. It is sub divided into five types. They are

S.No	Pitham	General Features	Changes in Kalladaippu
1	Anarpitham	Peps up the appetite and aids in digestion.	Normal
2	Ranjagapitham	Responsible for the colour and contents of blood.	Normal
3	Saathagapitham	Controls the whole body and is held responsible for fulfilling a purpose.	Affected (Dysuria, Oliguria)
4	Pirasagapitham	Dwells in the skin and concerned with the shine, glow, texture and its complexion.	Normal
5	Alosagapitham	Responsible for the perception of vision.[28]	Normal

KABHAM

It is responsible for the stream line functions of the body and maintains body's defence mechanism intact. It is again classified into 5 types.

S.No	KABHAM	GENERAL FEATURES	CHANGES IN KALLADAIPPU
1	Avalambagam	Lies in the respiratory organs, exercises authority over other kabhas and control the heart and circulatory system.	Normal
2	Kilethagam	Found in stomach as it seat, moistens the food, softens and helps to be digested.	Normal
3	Pothagam	Responsible for the perception of taste	Normal
4	Tharpagam	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid.	Normal
5	Santhigam	Necessary for the lubrication and the free movements of joints[29]	Normal

PARUVAKALAM

S.No	Perum pozhuthugal	Mukkutra marupaadugal
1	Kaar kaalam (Aavani & Purattasi) Mid August to Mid October	VATHAM - Vaetrunilai valarchi PITHAM – Thannilai valarchi
2	Koothir kaalam (Iypasi & Karthigai) Mid October to Mid December	VATHAM – Thannilai adaidhal PITHAM - Vaetrunilai valarchi
3	Munpani kaalam (Margazhi & Thai) Mid December to Mid February	PITHAM – Thannilai adaidhal
4	Pinpani kaalam (Masi & Panguni) Mid February to Mid June	KABHAM – Thannilai valarchi
5	Elavenir kaalam (Chithirai & Vaikaasi) Mid April to Mid June	KABHAM – Vaetrunilai valarchi
6	Mudhuvenir kaalam (Aani & Aadi) Mid June to Mid August	VATHAM – Thannilai valarchi KABHAM – Thannilai adaidhal[30]

THINAI (LAND)

Siddhars classified the lands into five types. They are

1. Kurunji – Mountain range
2. Mullai – Pastoral area of the forest
3. Marudham – The fertile river bed
4. Neidhal – The coastal region
5. Paalai – Arid desert

Kabha diseases will occur in Kurinji land. Pitha diseases occur in Mullai land. Vadha diseases occur in Neidhal land. Staying in Paalai land is not good to health. Marudham land is the fertile area where no disease occurs. So, Marudham land is the best one to stay.The winter season gives good health to the man, early summer and later rainy gives moderate health. Whereas early rainy and later summer are more prone to diseases, that's why siddhars called it as Aanaga kaalam. [31]

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINAIGAL

Mukkutram	**Paruvakaalam (Seasons)**			**Thinai**
	Thannilai valarchi (Accumulation)	**Vaetrunilai valarchi (Aggravation)**	**Thannilai adaidhal (Alleviation)**	
VATHAM	Mudhuvenil kaalam	Kaar kaalam	Koothir kaalam	Vatha disease is more prevalent in Neidhal land
PITHAM	Kaar kaalam	Koothir kaalam	Munpani kaalam	Pitha disease is more prevalent in Mullai land
KABHAM	Pinpani kaalam	Elavenil kaalam	Mudhuvenil kaalam[32]	Kabha disease is more prevalent in Kurunji land[33]

UDAL VANMAI (IMMUNITY)

Siddhars classify udal vanmai into three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

UDAL KATTUGAL

S.No	Udal kattugal	General Features	Changes in Kalladaippu
1	Saaram (Digestive essence)	Responsible for the growth and development. It keeps the individual in good temperament and it enriches the body.	Affected due to pain
2	Senneer (Blood)	Responsible for the color of the blood and for the intellect, nourishment, strength of the body.	Normal
3	Oon (Muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body.	Normal
4	Kozhuppu (Fat)	Lubricates the organs to facilitate frictionless functions.	Normal
5	Enbu (Bones)	Supports and protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body.	Normal
6	Moolai (Bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other system of body.	Normal
7	Sukkilam/Suronitham	Responsible for reproduction.[34]	Normal

PINIYARI MURAIMAI (DIAGNOSIS)

Four steps are followed in diagnosing the disease. They are

1. Poriyaal aridhal
2. Pulanal therdhal
3. Vinaadhal
4. Envagai thervugal

PORIYAAL ARIDHAL:

In this, the physician should carefully observe the changes that occur in the five sensory organs (porigal) of the patient.

PULANAL THERDHAL:

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

VINAADHAL:

The physician should interrogate about the patients name, age, occupation, socio-economic status, food habits, history of past illness, history of present illness, family history, marital status, menstrual history and frequency of pain.

ENVAGAI THERVUGAL:

“நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்.”[35]

Nowadays advanced diagnostic tools have been developed by modern bio medical scientists. But siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

Eight fold system of clinical assessments:

Siddhars have given eight diagnostic methodological tools. They are

1. Naadi
2. Sparisam
3. Naa
4. Niram
5. Mozhi
6. Vizhi
7. Malam
8. Moothiram[36]

GENERAL FINDINGS:

NAADI:

Naadi is responsible for the existence of life, can be felt one inch below the wrist on the radial side by means of palpation with tips of index, middle and ring finger, corresponding to vatham, pitham, kabham.

Three humours Vatham, Pitham, and Kabham are in the ratio 1:1/2:1/4 normally. Derangement in these ratio leads to various disease conditions.

Naadi nadai in kalladaippu

When the vatham add with mandham it produces the kalladaippu disease.

“ஏவலாய் குழலாய் பித்தஞ் செய்குணம் விளம்பக் கேளாய்
கோலவேல் விழி சிவந்து குளிர்ந்திடிரிருக்கு மல்லால்
சீலவே நீர்கடுத்து நொந்து சுறுக்கெனச் சிவந்து வீழும்
ஞலமே கிறுகிறென்று நாவுலர்ந் திருக்குங் காணே.”[37]

SPARISAM:

By sparisam, the temperature of skin (thatpam- cold or veppam – heat), smoothness, roughness, sweating, dryness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In Kalladaippu, patient feels tenderness over the lower abdomen, renal angle and lumbar region. Also patient's temperature is increased in lower abdomen, sweating all over the body at the time of colic.

NAA:

Signs and symptoms in the tongue are noted here. Colour, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

- In kalladaippu, the naa is not affected.

NIRAM:

The colour of the skin is noted here.

- In kalladaippu, the Niram may be affected in sukkila achmari.

MOZHI:

Character of the speech is noted, mainly uraththa oli (high pitched), thazhndha oli (low pitched), or resembles the sound of any instrument.

- In kalladaippu, the mozhi will be affected to the patients who have severe pain leading to the thazhndha oli.

VIZHI:

Character of the eye is noted. Colour, warm, burning sensation, irritation, visual perception are generally noted.

- In kalladaippu, the vizhi may be affected. Redness due to renal colic pain.

MALAM:

The stools are examined for quantity, hardening (malakattu), loose motion (bedhi), colour and smell.

- In kalladaippu, the malam will be affected due to either constipation or diarrhoea.

MOOTHIRAM

a) NEERKURI (Urine examination)

Urine examination is good diagnostic method compare to naadi and other Envagai thervugal. Theraiyar mention it as.

"அருந்து மாறி ரதமும் அவிரோதமமாய்
அக்கல் அலர்தல் அகாலவூன் தவிர்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காதுபெய்
தொருமுகூர்த்தக் கலைக்குட்பட்டு நீரின்
நிறக்குறி நெய்குறி நிருமித்தல் கடனே."[38]

The early morning urine sample is collected and sample should be examined within one and hour hours.

SIRUNEERIN POTHU GUNAM:

"வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென
றைந்தியலுளவை யறைகுது முறையே."[38]

The urine is examined for its Niram (colour), Eadai (Specific gravity), Nurai (Froth), Natram (Smell), Enjal (Deposits). In kalladaippu, the moothiram is affected due to scanty Micturition.

NIRAM (COLOUR)

"பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதைங் கொழுமையை யொத்துகு நீரே."[39]

1. Yellow
2. Red
3. Green
4. Black
5. White

KALLADAIPPU NEERIN GUNAM (COLOUR INDICATING URINARY STONES)

The urine colour would look like flesh washing water; this is indicated in kidney diseases. This is mentioned as

"தீப்புலால் கழுநீர்ச் செயலெனிர் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறகப்
பற்பகலாகப் பையப் பதிந்தே."[40]

EADAI (SPECIFIC GRAVITY)

Urine, not thick is considerably healthy. This is mentioned as

"மிகத் தடிப்பும் மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும் மெய்ச் சுபாவ நீர் நன்றே."[41]

NURAI (FROTH)

Urine may be frothy in nature, if it is reduced in vali, azhal and ayyam are said to be deranged. This is mentioned as

"பந்தமெய்ப் பசையிளகப்படும் பருவத்
தந்தர்ப் பூதமாய் அனில மூத்திரத்தில்
சம்பத்தப்படும் ததிநுரைப் புனலே."[42]

NAATRAM (SMELL)

Foul odour with pyuria is observed in patients with urinary lithiasis associated with urinary tract infection and ulcer. This is mentioned as

“ஓதமணத்தோ டவவோத மொத்தி றங்கும்
சீதளத்தாற் கம்மிய தேகிகளுக்கே
காணிதில சீழுற் கலந்திழி மணமுறின்
கருப்பநா பிகளுங் காமநா ளத்துளும்
விரணமுண் டின்றேல் எய்து மாசுமரியல்
திருத்தலே திண்ண மெனமனத் துன்னே.”[41]

ENJAL (DEPOSITS)

If urine excretion look like curd water white colour and sand like deposits in urine indicate stones in kidney. This is mentioned as

“நார்த்தி நீர்ப்பால் போல நனவுற்றங் கிழியு மானால்
மாரற்ப முற்ற நீரி லடி மண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய் பற்றிய கல்வி னாலே
சீருற்ற செய்கை யென்று தெரிவுறச் செப்ப லாமே.”[43]

NEIKURI

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

The urine kept on the kidney tray in sun light, on non wind condition, should be by dropping a drop of gin oil gently with rod. If oil spread like snake, it indicates vali neer; a ring indicates azhal neer and float like a pearl indicates iyya neer and sinks in urine indicates mukkutram.

"அரவென நீண்டினஃதே வாதம்

ஆழி போல் பரவின் அஃதே பித்தம்

முத்தொத்து நிற்கின் மொழிவதென் கபமே".[44]

- Vatha neer – The oil spreads like snake
- Pitha neer – The oil spreads like ring
- Kabha neer – The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis[45]
- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis.[46]

Since kalladaippu is due to the derangement of vatham and pitham, the Neikuri will be vatha or pitha neer.

MARUTHUVAM (LINE OF TREATMENT)

The entire siddha system of medicine consists of three great subdivisions namely,

1) Noyillaneri (preventive) – Kaappu
2) Noineekuneri (curative methods) – Neekkam
3) Uramaakkumurai (strengthening methods) - Niraippu .

Noyillaneri is the special approach of the siddha system where regular dietary habits, early rising, physical and mental discipilinaries are all emphasized. Prevention can mostly save our body and soul, but modernization results in alteration of good health, leads to disease.

Siddha system is playing major role in treating and preventing many chronic diseases. Likewise, herbal medicines have several phyto chemicals which exert their beneficial effect on urolithiasis by multiple mechanisms like,

- Diuretic activity
- Crystallization inhibiting activity
- Lithotriptic activity

- Antimicrobial activity
- Analgesic and anti inflammatory activity
- Improving renal function
- Regulates oxalate, calcium mechanisms.

The main object of treatment is to bring down the deranged mukkutrams to natural equilibrium by giving purgatives, which cure derangement of vatham; this is one of the causes for kalladaippu.

In Siddha system, treatment is not only removable of disease but also the prevention and improving the body condition after removal of disease. This is said as kappu, neekkam and niraippu.

Fomentation:

An attack of renal colic may be aborted by the application of heat fomentation (hot water bottle or heater) to the lumbar region. Immediate treatment of loin pain or renal colic is bed rest.

PREVENTION:

1. For prophylactic purpose it is necessary to eliminate all hindrances to a free drainage of urine (constriction, adenoma of the prostrate etc) and to remove foci of infection from the teeth and tonsils.
2. To prevent the formation of urate calculi, a diet of milk and vegetables and mineral water is prescribed.
    a. In the presence of oxalate calculi restrictions are imposed on foods rich in calcium (milk, raw eggs, potatoes) with total abstinence from chocolate, spinach gooseberries and carrots.
3. A patient with phosphorus, carbonate stones is kept on a meat diet and much water to drink.

ADVICE:

1. Patients should drink large amount of water (2 - 3 lit/day)
2. Patient should not suppress the excretion of urine and seminal fluid.
3. Preparation containing Vit. D must be avoided.
4. Regarding prevention Anubhava vaidhya deva ragasiyam states that one should not suppress the excretion of Moothiram (urine) and Sukkilam (Seminal fluid).

NOI KANIPPU VIVADHAM (DIFFERENTIAL DIAGNOSIS)

1) Neerkattu (Anuria)
2) Neerchurukku (Oliguria)
3) Chottu neer (Incontinence)

DO'S AND DONT'S:

DO'S:

1) Drink 2-3 litres of water per day.
2) Dring tender coconut, barley water, lemon juice, raddish juice.
3) The following vegetables can be taken in the diet

   Raddish

   Lady's finger

   Plantain pith

   Mint leaves

   Bottle guard

DONT'S:

1) Avoid cabbage, cauliflower, and tomato seeds, mushroom.
2) Avoid milk and its products.
3) Avoid chicken, fish and other sea foods

   Avoid drinking fluoride containing water

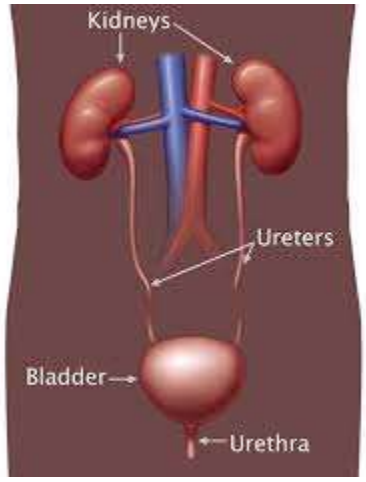
MODERN ASPECT

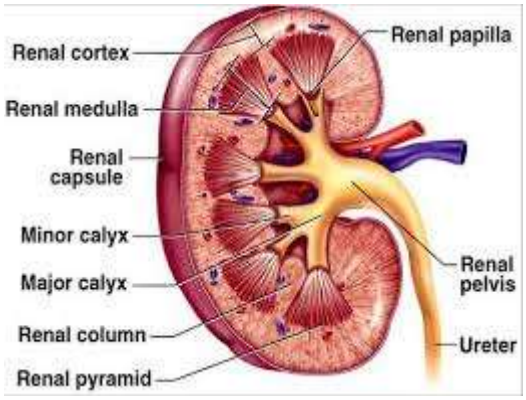
MODERN ASPECT

ANATOMY AND PHYSIOLOGY OF THE URINARY SYSTEM

KIDNEYS

The kidneys are a pair of excretory organs situated on the posterior abdominal wall, one on each side of the vertebral column, behind the peritoneum. They remove waste products of metabolism and excess of water and salts from the blood, and maintain its pH.





EXTERNAL FEATURES

Each kidney is bean shaped. It has upper and lower poles, medial and lateral borders, and anterior and posterior surfaces.

TWO POLES OF THE KIDNEY

The upper pole is broad and is in close contact with the corresponding suprarenal gland. The lower pole is pointed.

TWO SURFACES

The anterior surface is said to be irregular and the posterior surface flat, but it is often difficult to recognize the anterior and posterior aspects of the kidney by looking at

the surfaces. The proper way to do this is to examine the structures present in the hilum as described below.

TWO BORDERS

The lateral border is convex. The medial border is concave. Its middle part shows a depression, the hilus or hilum.

HILUM

The following structures are seen in the hilum from anterior to posterior side: 1) The renal vein 2) the renal artery and 3) the renal pelvis, which is the expanded upper end of the ureter. Examination of these structures enables the anterior and posterior aspects of the kidney to be distinguished from each other. As the pelvis is continuous, inferiorly, with the ureter the superior and inferior poles of the kidney can also be distinguished by examing the hilum. So it is possible to determine the side to which a kidney belongs by examining the structures in the hilum. Commonly, one of the branches of the renal artery enters the hilus behind the renal pelvis, and a tributary of the renal vein may be found in the same plane.

LOCATION

The kidneys occupy the Epigastric, hypochondriac, lumbar and umbilical regions. Vertically they extend from the upper border of twelfth thoracic vertebra to the centre of the body of the third lumbar vertebra.

The right kidney is slightly lower than the left, and the left kidney is a little nearer to the median plane than the right.

SHAPE, SIZE, WEIGHT AND ORIENTATION

Each kidney is about 11cm long, 6 cm broad and 3 cm thick. The left kidney is a little longer and narrower than the right kidney. On an average the kidney weighs 150 g in males and 135 g in females. The kidneys are reddish brown in colour.

The long axis of the kidney is directed downwards and laterally, so that the upper poles are nearer to the median plane than the lower poles. The transverse axis is directed laterally and backwards.

CAPSULES OR COVERINGS OF KIDNEY

1. **The fibrous capsule**: This is a thin membrane which closely invests the kidney and lines the renal sinus.
2. **Perirenal or perinephric fat**: This is a layer of adipose tissue lying outside the fibrous capsule. It is thickest at the borders of the kidney and fills up the extra space in the renal sinus.
3. **Renal fascia:** This is a fibroareolar sheath which surrounds the kidney and the perirenal fat called as the fascia of Gerota. It consists of an anterior layer or fascia of Toldt and a posterior layer or fascia of Zuckerkandl.
4. **Pararenal or paranephric body (fat**): It consists of a variable amount of fat lying outside the renal fascia. It is more abundant posteriorly and towards the lower pole of the kidney. It fills up the paravertebral gutter and forms a cushion for the kidney.

structure

Naked eye examination of a coronal section of the kidney shows: a) an outer, reddish brown cortex; b) an inner, pale medulla; c) a space, the renal sinus.

The renal medulla is made up of about 10 conical masses, called the renal pyramids. Their apices form the renal papillae which indent the minor calices.

The renal cortex is divisible into two parts: a) cortical arches or cortical lobules, which form caps over the bases of the pyramids; and b) renal columns, which dip in between the pyramids. Each pyramid along with the overlying cortical arch forms a lobe of the kidney.

The renal sinus is a space that extends into the kidney from the hilus. It contains a) branches of the renal artery; b) tributaries of the renal vein; and c) the renal pelvis. The

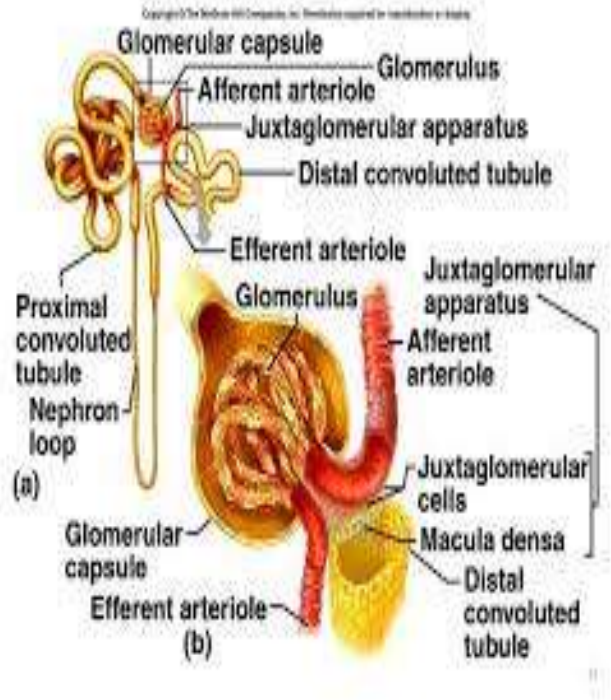
pelvis divides into 2 to 3 major calices, and these in their turn divide into 7 to 13 minor calices. Each minor calyx ends in an expansion which is indented by one or three renal papillae.

Histologically, each kidney is composed of one to three million uriniferous tubules. Each tbule consists of two parts which are embryologically distinct from each other. These are as follows.

A) The **secretory part**, called the nephron, which elaborates urine. Nephron is the functional unit of kidney and comprises the renal corpuscle or Malphigian corpuscle and the renal tubule.

B) The **collecting tubule** begins as a junctional tubule from the distal convoluted tubule. Many tubules unite together to form the ducts of Bellini which open into minor calices through the renal papillae.

C) Juxtaglomerular apparatus is formed at the vascular pole of glomerulus which is intimately related to its own ascending limb of the Henle's loop near the distal convoluted tubule.



BLOOD SUPPLY

ARTERIAL SUPPLY

Usually there is one renal artery on each side, arising from the abdominal aorta. Accessory renal arteries are present in 30% of individuals; they arise commonly from the aorta, run parallel to the renal artery, and enter the kidney either at the hilus or at one of its poles.

At or near the hilus the renal artery divides into anterior and posterior divisions. Further branching of these divisions gives rise to segmental arteries each of which supplies one vascular segment. Five such segments are described. These are apical, upper, middle, lower and posterior. The segmental arteries are end arteries, so that the vascular segments are independent units.

VENOUS DRAINAGE

The venous end of the peritubular capillary plexus gives rise to interlobular veins which run along the corresponding arteries. The interlobular veins drain into the arcuate veins, which in their turn open into the interlobar veins. These emerge at the renal sinus and join to form the renal vein which drains into the inferior vena cava.

The venous end of the capillary plexus along the vasa recta gives rise to veins which drain into the arcuate veins.

LYMPHATIC DRAINAGE

The lymphatics of the kidney drain into the lateral aortic nodes located at the level of origin of the renal arteries.

NERVE SUPPLY

The kidney is supplied by the renal plexus, an offshoot of the celiac plexus. It contains sympathetic (T10 – L1) fibres which are chiefly vasomotor. The afferent nerves of the kidney belong to segments T10 to T12.[47]

FUNCTIONS OF KIDNEYS

Kidneys perform vital functions. By excreting urine, kidneys play principal role in the maintenance of internal environment. In addition, kidneys perform many other functions as described below.

1.ROLE OF HOMEOSTASIS

The primary function of kidneys is homeostasis. It is accomplished by the formation of urine. Kidneys are not only the excretory organs, but are also the regulatory organs because their major role is in homeostasis. During the formation of urine, kidneys regulate various activities in the body, which are concerned with homeostasis.

Excretion of waste products: Removal of wastes help in homeostasis. Kidneys excrete the unwanted waste products which are formed during metabolic activities.

a. Urea – end product of amino acid metabolism
b. Uric acid – end product of nucleic acid metabolism
c. Creatinine – end product of metabolism in muscles
d. Bilirubin – end product of hemoglobin degradation
e. Products of metabolism of other substances.

Kidneys also excrete harmful foreign chemical substances like:

a. Toxins
b. Drugs
c. Heavy metals
d. Pesticides etc.,

Maintenance of water balance: Kidneys maintain the water balance in the body by conserving water when it is decreased and excreting water when it is excess in the body. This is a very important process for homeostasis.

Maintenance of electrolyte balance: Maintenance of electrolyte balance, especially sodium is in relation to water balance. Kidneys retain sodium if the osmolarity of body water decreases and eliminate sodium when osmolarity increases.

Maintenance of acid base balance: The pH of the blood and body fluids should be maintained within narrow range for healthy living. It is achieved by role of kidneys. Body is under constant threat to develop acidosis, because of production of lot of acids during metabolic activities. However, it is prevented by kidneys, lungs and blood buffers, which eliminate these acids. Among these organs, kidneys play major role in preventing acidosis. In fact, kidneys are the only organs, which are capable of eliminating certain metabolic acids like sulfuric and phosphoric acids.

2. HEMOPOIETIC FUNCTION

Kidneys stimulate the production of erythrocytes by secreting erythropoietin. Erythropoietin is the important stimulating factor for erythropoiesis. Kidney also secretes another factor called thrombopoietin, which stimulates the production of thrombocytes.

3.ENDOCRINE FUNCTION

Kidneys secrete many hormonal substances in addition to erythropoietin and thrombopoietin. The hormones secreted by kidneys are:

a. Erythropoietin
b. Thrombopoietin
c. Renin
d. 1, 25 – Dihydroxycholecalciferol
e. Prostaglandins

4.REGULATION OF BLOOD PRESSURE

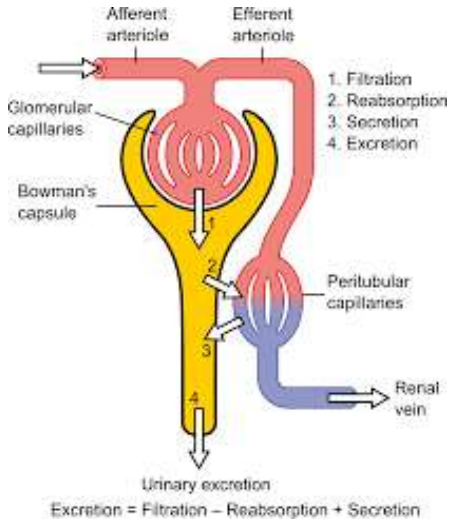
Kidneys play an important role in the regulation of arterial blood pressure. Kidneys regulate arterial blood pressure by two ways:

1. By regulating the volume of extracellular fluid
2. Through renin – angiotensin mechanism

5.REGULATION OF BLOOD CALCIUM LEVEL

Kidneys play a role in the regulation of blood calcium level by activating 1, 25 – dihydroxycholecalciferol into vitamin D. Vitamin D is necessary for the absorption of calcium from intestine.[48]

MECHANISM OF URINE FORMATION



The process involve in urine formation are,

1. Glomerular filtration
2. Tubular reabsorption
3. Tubular secretion

GLOMERULAR FILTRATION

Glomerular filtrate is protein free plasma. Glomerular filtration is depends upon hydrostatic pressure of the afferent arterioles, glomerular capillary pressure and colloidal osmotic pressure. The glomerular filter contains all the substance present in the plasma except colloids.

Normal amount of urine excreted per day is about 1.5 litres. The glomerular filtrate is alkaline. It contains water, small quantities of urea, glucose, potassium, calcium, bicarbonates and uric acid.

TUBULAR REABSORPTION

When the glomerular filtrate flows through the tubular portion of nephron, both quantitive and qualitative changes occur. Large quantity of water (more than 99%), electrolytes and other substances are reabsorbed by the tubular epithelial cells. The

reabsorbed substances move into the interstitial fluid of renal medulla. And, from here, the substances move into the blood in peritubular capillaries.

Since the substances are taken back into the blood from the glomerular filtrate, the entire process is called tubular reabsorption.

TUBULAR SECRETION

In addition to reabsorption from renal tubules, some substances are also secreted into the lumen from the peritubular capillaries through the tubular epithelial cells. It is known as tubular secretion or tubular excretion.

Thus, urine is formed in the nephron by the processes of glomerular filtration, selective reabsorption and tubular secretion.[49]

URETERS

The ureters are a pair of narrow, thick-walled muscular tubes which convey urine from the kidneys to the urinary bladder.

They lie deep to the peritoneum, closely applied to the posterior abdominal wall in the upper part, and to the lateral pelvic wall in the lower part.

DIMENSIONS

Each ureter is about 25 cm (10 in.) long, of which the upper half (5 in.) lies in the abdomen, and the lower half (5 in.) in the pelvis. It measures about 3mm in diameter, but it is slightly constricted at three places.

COURSE

The ureter begins within the renal sinus as a funnel shaped dilatation, called the renal pelvis. The pelvis issues from the hilus of the kidney, descends along its median margin, or partly behind it. Gradually it narrows till at the lower end of the kidney it becomes the ureter proper.

The ureter passes downwards and slightly medially on the psoas major muscle, and enters the pelvis by crossing in front of the termination of the common iliac artery. In the lesser or true pelvis the ureter at first runs downwards, and slightly backwards and

laterally, following the anterior margin of the greater sciatic notch. Opposite the ischial spine it turns forwards and medially to reach the base of the urinary bladder. The ureter enters the bladder wall obliquely to open into it at the lateral angle of its trigone.

CONSTRICTIONS

The ureter is slightly constricted at three places: 1) at the pelviureteric junction; 2) at the brim of the lesser pelvis; 3) at its passage through the bladder wall. The renal stones tend to get arrested at these places.

BLOOD SUPPLY

Upper part receives branches from renal artery, gonadal or colic vessels, middle part receives branches from aorta, the gonadal or iliac vessels, and pelvic part is supplied by branches from the vesical, middle rectal or uterine vessels.

NERVE SUPPLY

The ureter is supplied by sympathetic from T10 – L1 segments and parasympathetic from S2 – S4 nerves. They reach the ureter though the renal, aortic and hypogastric plexuses. All the nerves appear to be sensory in function.[50]

URINARY BLADDER

The urinary bladder is a muscular reservoir of urine, which lies in the anterior part of the pelvic cavity. The detrusor muscle of urinary bladder is arranged in whorls and spirals and is adapted for mass contraction rather than peristalsis.

SIZE, SHAPE AND POSITION

The bladder varies in its size, shape and position according to the amount of urine it contains. When empty it lies entirely within the pelvis; but as it fills it expands and extends upwards into the abdominal cavity, reaching up to the umbilicus or even higher.

EXTERNAL FEATURES

An empty bladder is tetrahedral in shape and has: a) An apex, directed forwards; b) a base or fundus, directed backwards; c) a neck, which is the lowest and most fixed part of the bladder; d) three surfaces, superior and right and left inferolateral; and e) four borders, two lateral, one anterior and one posterior.

A full bladder is ovoid in shape and has: a) An apex, directed upwards towards the umbilicus; b) a neck, directed downwards, and c) two surfaces, anterior and posterior.

INTERNAL SPHINCTER OF THE BLADDER

The bladder wall is made up of longitudinal and circular layers of smooth muscles and they are called detrusor muscle. In the trigone in addition to detrusor muscle, there is trigonal muscle of bell. There is no definite circular muscle fibre at the neck of the bladder stop at the level of neck. Longitudinal fibres from the posterior wall diverge to pass around the urethra on both sides.

CAPACITY OF THE BLADDER

The mean capacity of the bladder in an adult male is 220 ml, varying from 120 to 320 ml. filling beyond 220 ml causes a desire to micturate, and the bladder is usually emptied when filled to about 250 – 300 ml. filling upto 500 ml may be tolerated, but beyond this it becomes painful. Referred pain is felt in the lower part of the anterior abdominal wall, perineum and penis (T11 to L2; S2 – S4).

BLOOD SUPPLY

Superior vesicle arteries and inferior vesicle arteries supplies the bladder. In addition branches from obturator and inferior gluteal artery are supplied to the bladder.

NERVE SUPPLY

Sympathetic fibers arise from T11 – L2 segment. Parasympathetic fibres branches from S2 – S4. Somatic pudental nerve supplies the sphincter urethrae which is voluntary.[51]

URETHRA

Urethra is a tubular passage extending from the neck of the bladder to the external urethral orifice.

The male urethra extends from the internal urethral orifice at the neck of urinary bladder to the external urethral orifice at the tip of the penis. It is about 20 cm long in flaccid state of the penis; the long axis of urethra shows 2 curvatures and is therefore S shaped. In the erect state it becomes J shaped.

It is divided into 3 parts

1) Prostatic part : Passes through prostate (3 cm long)
2) Membraneous part : Surrounded by sphincter (2 cm long)
3) Spongy part (Penile part) : Passes through the bulb and carpus spongiosum (15 cm long) [52]

SPHINCTER OF THE URETHRA

There are 2 sphincters, in relation with urethra internal and external. The internal sphincter made up of smooth muscle fibre and situated at the neck of the bladder is supplied by sympathetic nerves from lower thoracic segments and upper lumbar segments.

The external sphincter made of light striated muscle fibre surrounds the membraneous part of urethra; it is supplied by prineal branch of the pudental nerve (S2 to S4).

BLOOD SUPPLY

Branches of internal pudental artery supplies urethra. [53]

THE FEMALE URETHRA

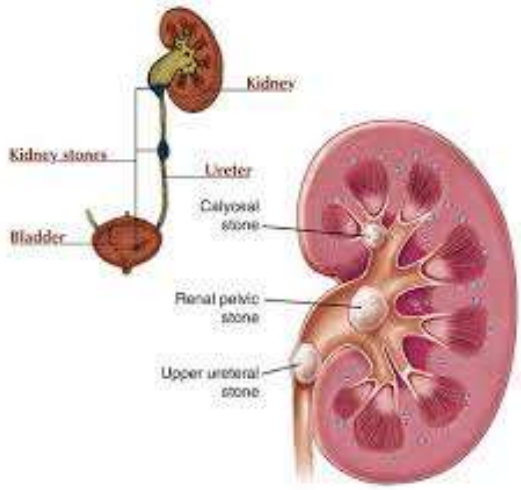
The female urethra is only 4 cm long and 6 mm in diameter. Developmentally, it corresponds to the upper part of the prostatic urethra of the male.

It begins at the internal urethral orifices roughly 5 cm behind the middle of the pubic symphysis. It runs downwards and forwards embedded in the anterior wall of the vagina, traverse the urogenital diaphragm and ends at the lateral urethral orifices in the vestibule.

The mucosa of the urethra is much folded and contains numerous mucous glands and lacunae which open into the urethra. The collections of mucous glands one on each side of the upper part of the urethra is called the paraurethral glands of skene.

The female urethra is dilatable.[53]

RENAL CALCULI (UROLITHIASIS)



It refers to calculus formation at any level in the urinary tract but most arise in the kidney.calculi are common in the renal pelvis, calyces and collecting ducts of people in industrialized countries, and is the third most common disorder of the urinary tract. [54]

Urolithiasis is a frequent clinical problem, affecting 5 to 10% of Americans in theirlifetime. Males are affected more often than females and the peak age at onset is between20 and 30 years. Familial hereditary predisposition to stone formation has long been known. Many of the inborn errors of metabolism, such as gout cystinuria, and primaryhyperoxaluria, provide good examples of hereditary disease characterized byexcessiveproduction and excretion of stone -forming substances.[55]

In underdeveloped countries nephrolithiasis is rare. In Britain and USA the incidence of renal calculi has become at least 10 times higher in the past 90 years. This increase may be related to changes in diet e.g. increase in protein diet. Current annual incidence in Britain and the USA ranges from 6.87 to 20.8 per 10,000 of the population. A familial tendency toward stone formation has long been recognized

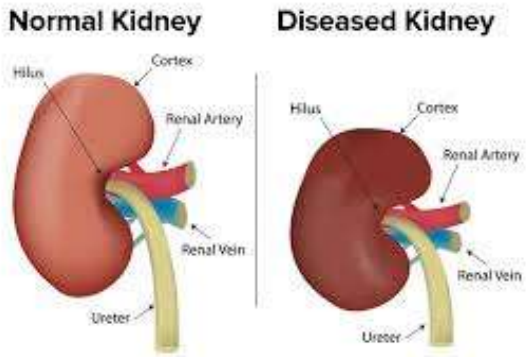
If stones grow to sufficient size (usually at least 3 millimeters (0.12 in)) they can cause obstruction of the ureter. Ureteral obstruction causes postrenal azotemia and hydronephrosis (distention and dilation of the renal pelvis and calyces), as well as spasm of the ureter. This leads to pain, most commonly felt in the flank (the area between the

ribs and hip), lower abdomen, and groin (a condition called renal colic). Renal colic can be associated with nausea, vomiting, fever, blood in the urine, pus in the urine, and painful urination. Renal colic typically comes in waves lasting 20 to 60 minutes, beginning in the flank or lower back and often radiating to the groin or genitals. The diagnosis of kidney stones is made on the basis of information obtained from the history, physical examination, urinalysis and radiographic studies. Ultrasound examination and blood tests may also aid in the diagnosis.[56]

Renal calculi are formed when the urine is supersaturated with salt and minerals such as calcium oxalate, struvite (ammonium magnesium phosphate), uric acid and cystine.60-80% of stones contain calcium[57]. They vary considerably in size from small 'gravel-like' stones, to large staghorn calculi. The calculi may stay in the position in which they are formed, or migrate down the urinary tract, producing symptoms along the way. Studies suggest that the initial factor involved in the formation of a stone may be the presence of nanobacteria that form a calcium phosphate shell[58,59.].

The other factor that leads to stone production is the formation of Randall's plaques. Calcium oxalate precipitates form in the basement membrane of the thin loops of Henle; these eventually accumulate in the subepithelial space of the renal papillae, leading to a Randall's plaque and eventually a calculus.[60]

Pathophysiology



Urinary calculi are consist of aggregates of crystals, usually containing calcium or phosphate in combination with small amounts of proteins and glycoproteins. In developed countries, however, most calculi occur in healthy young men, in whom investigations reveal no clear predisposing cause. Renal stones vary greatly in size. There may be particles like sand anywhere in the urinary tract, or large round stones in the bladder.

In developing countries bladder stones are common, particularly in children. In developed countries, the incidence of childhood bladder stones is low; renal stones in adults are more common. Staghorn calculi fill the whole renal pelvis and branch into the calyces; they are usually associated with infection and composed largely of struvite.

Deposits of calcium may be present throughout the renal parenchyma, giving rise to fine calcification within it (nephrocalcinosis), especially in patients with renal tubular acidosis, hyperparathyroidism, vitamin D intoxication and healed renal tuberculosis. Cortical nephro calcinosis may occur in areas of cortical necrosis, typically after AKI in pregnancy or other severe AKI.

PREVALENCE

Renal stone disease is common , affecting individuals of all countries and ethnic groups. In the UK, the prevalence is about 1.2%, with a lifetime risk of developing a renal stone at age 60-70% of about 7% in men. In some regions the risk is higher, most notably in countries like Saudi Arabia, where the lifetime risk of developing a renal stone in men aged 60-70 is just over 20%[61].

EPIDEMIOLOGY

- Renal stones are common, being present at some time in one in ten of the population, although a significant proportion will remain asymptomatic.
- The annual incidence is about 1-2 cases of acute renal colic per 1,000 people and the average lifetime risk around 5-10%.

- Men are more commonly affected than women, with a male to female ratio of 3:1. The difference between the sexes is gradually being eroded. This is thought to be due to lifestyle-associated factors, such as obesity and a Western diet.
- The peak age for developing stones is between 30 and 50 and recurrence is common[57].

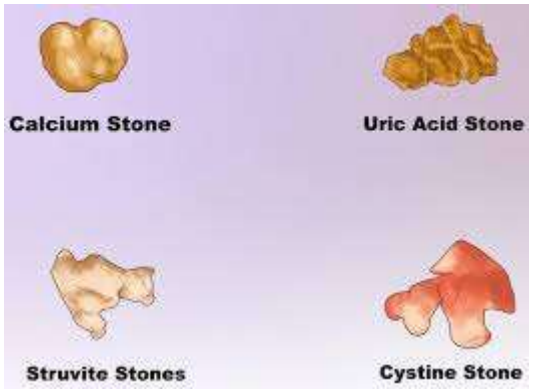
ETIOLOGY AND PATHOGENESIS

The crystals in the stone in the kidney are commonly mixed, although there is usually a preponderance of crystals from one particular solute. 70-75% of renal stones are composed almost entirely of calcium oxalate, mixed with calcium phosphate. 15% of calculi consist predominantly of magnesium ammonium phosphate (struvite). Uric acid stones and cysteine stones together account for about 10% of calculi. Very rarely stones are made up of almost pure xanthine, silica or some chemical foreign to the body.

Uric acid crystals are commoner in some parts of the Middle East, India and North Africa than in Europe and the USA.

The formation of stones within the kidney is not a specific disease, it is potential complication of many different disorders. The cause is obscure. The most important is almost certainly an increased urine concentration of the some constiuents[62].

TYPES OF RENAL CALCULI



There are 4 main tyoes of renal calculi- calcium containing, mixed(struvite), uric acid and cysteine stones, a few rare types.

1) CALCIUM STONES

Calcium stones are the most common comprising about 75% of all urinary calculi. They may be pure stones of calcium oxalate (50%), or calcium phosphate (5%), or mixture of calcium oxalate and calcium phosphate (45%).

Etiology

Etiology of calcium stones are variable.

About 50% of patients with calcium stones have *idiopathic hypercalciuria without hypercalcaemia.*

Approximately 10 % cases are associated with *hypercalcaemia and hypercalciuria*, the most commonly due to hyperparathyroidism, or a defect in the bowel (i.e. absorptive hypercalciuria), or in the kidney (i.e. renal hypercalciuria).

About 15% of patients with calcium stones have *hyperuricosuria with a normal blood uric acid level* and without any abnormality of calcium metabolism.

In about 25% of patients with clcium stones, the cause is known as there is no abnormality in urinary excretion of calcium, uric acid or oxalate and is referred to as *idiopathic calcium stone disease*.

Pathogenesis

The mechanism of calcium stone formation is explained on the basis of imbalance between the degree of supersaturation of the ions forming the stone and the concentration of inhibitors in the urine. Most likely site where the crystals of calcium oxalate and / or calcium phosphate are precipitated is the tubular lining or around some fragment of debris in the tubule acting as nidus of the stone. The predisposing factors contributing to formation of calcium stones are alkaline pH, decreased urinary volume and increased excretion of oxalate and uric acid

Morphology

Calcium stones are usually small (less than a centimeter), ovaid, hard, with granular rough surface. They are dark brown due to old blood pigment deposited in them as a result of repeated trauma caused to the urinary tract by these sharp – edged stones.

2.MIXED STONES

About 15% of urinary calculi are made of magnesium-ammonium-calcium phosphate, often called struvite; hence mixed stones are also called as *struvite stones* or triple phosphate stones.

Etiology

Struvite stones are formed as a result of infection of the urinary tract with urea- splitting organisms that produce urease such as by specious of Proteus, and occasionally klebsiella, pseudomonas, and Enterobacter. these are, therefore, also known as infection – induced stones. However, E.coli does not form urease.

Morphology

Struvite stones are yellow- white or grey. They tend to be soft and irregular in shape. Staghorn stone which is large, solitary stone that takes the shape of the renal pelvis where it is often formed is an example of struvite stone[63]

3).URIC ACID STONES

Approximately 6% of urinary calculi are made of uric acid. Uric acid calculi are *radiolucent* unlike radio- opaque calcium stones.

Etiology

Uric acid stones are frequently formed in cases with hyperuricaemia and hyperuricosuria such as due to primary gout or secondary gout due to myeloproliferative disorders (e.g. in leukaemias), especially those on chemotherapy, and administration of uricosuric drugs (e.g. salicylates, probenacid). Other factors contributing to their formation are acidic urinary pH (below 6) and low urine volume.

Pathogenesis

The solubility of uric acid at pH of 7 is 200 mg/ dl while at pH of 5 is 15 mg /dl. Thus as the urine becomes more acidic, the solubility of uric acid in urine decreases and precipitation of uric acid crystals increases favoring the formation of uric acid stones.

Hyperuricosuria is the most important factor in the production of uric acid stones, while hyperuricaemia is found in about half the cases.

Morphology

Uric acid stones are smooth, yellowish- brown, hard and often multiple. On cut section, they show laminated structure.

4).CYSTINE STONES

Cysteine stones comprise less than 2% of urinary calculi.

Etiology

Cysteine are associated with cystinuria due to a genitically- determined defect in the transport of cysteine and other amino acids across the cell membrane of the renal tubules and the small intestinal mucosa.

Pathogenesis

The resultant excessive excretion of cysteine which is least soluble of the naturally-occurring amino acids leads to formation of crystals and eventually cysteine calculi.

Morphology

cysteine stones are small, rounded, smooth and multiple. They are yellowish and waxy.

5.OTHER CALCULI

Less than 2% of urinary calculi consist of other rare types such as due to inherited abnormality of enzyme metabolism e.g. hereditary xanthinuria developing xanthine stones[64].

CLINICAL COURSE

Stones are of importance when they obstruct urinary flow or produce ulceration and bleeding. They may be present without producing any symptoms or significant renal damage. In general, smaller stones are most hazardous, as they may pass into the ureters, producing pain referred to as colic (one of the most intense forms of pain) as well as ureteral obstruction. Larger stones cannot enter the ureters and are more likely to remain silent within the renal pelvis. Commonly, these larger stones first manifest themselves by hematuria. Stones also predispose to superimposed infection, both by their obstructive nature and by the trauma they produce[65].

MORPHOLOGY OF KIDNEY

In 80% of patients, stones are unilateral. Common sites are renal pelvis, calyces and the bladder. Calcium oxalate crystals are mostly unilateral and solitary. They are usually either yellow-brown or dark from altered blood, and hard. Predominantly oxalate calculi may be nodular with smaller blunt spikes; mixed oxalate and phosphate stones may be fairly smooth. If small they are triangular in section. The nodular form may be called mulberry type in Britain or the jackstone type in the USA. Occasionally progressive

accretion of salts leads to the development of branching structures known as staghorn calculi, which create a cast of the renal pelvic and calyceal system. These massive stones are usually composed of magnesium ammonium phosphates[62].

CLINICAL FEATURES

The clinical presentation is highly variable. Most patients with renal stone disease are asymptomatic, whereas others present with pain, hematuria, UTI, or urinary tract infection.

A common presentation is with acute loin pain radiating to the anterior abdominal wall, together with hematuria; a symptom complex termed renal or ureteric calculi. This most commonly caused by a calculus but the same symptoms can occur in association with a sloughed renal papilla, tumour or blood clot.

The patient is suddenly aware of pain in the loin, which radiates round the flank to the groin and often into the testis or labium, in the sensory distribution of the first lumbar nerve. The pain steadily increase in intensity to reach a peak in a few minutes.

The patient is restless and generally tries unsuccessfully to obtain relief by changing position or packing the room. There is pallor, sweating, and often vomiting. Frequency, dysuria, and hematuria may occur. The intense pain usually subsides within 2 hours but may continue unabated for hours or days[61].

PRESENTATION

- Many stones are asymptomatic and discovered during investigations for other conditions.
- The classical features of renal colic (or ureteric colic) are sudden severe pain. It is usually caused by stones in the kidney, renal pelvis or ureter, causing dilatation, stretching and spasm of the ureter. In most cases no cause is found:

- Pain starts in the loin about the level of the costovertebral angle (but sometimes lower) and moves to the groin, with tenderness of the loin or renal angle, sometimes with haematuria.
- If the stone is high and distends the renal capsule then pain will be in the flank but as it moves down pain will move anteriorly and down towards the groin.
- A stone that is moving is often more painful than a stone that is static.
- The pain radiates down to the testis, scrotum, labia or anterior thigh.
- Whereas the pain of biliary or intestinal colic is intermittent, the pain of renal colic is more constant but there are often periods of relief or just a dull ache before it returns. The pain may change as the stone moves. The patient is often able to point to the place of maximal pain and this has a good correlation with the current site of the stone.

Other symptoms which may be present include:

- ❖ Rigors and fever.
- ❖ Dysuria.
- ❖ Haematuria.
- ❖ Urinary retention.
- ❖ Nausea and vomiting[57].

PREDISPOSING FACTORS FOR KIDNEY STONES

Environmental and dietary:

- Low urine volumes, high ambient temperatures, low fluid intake
- Diet: high protein intake, high sodium, low calcium
- High sodium excretion
- High urate excretion
- High oxalate excretion
- Low citrate excretion

Acquired causes:

- Hypercalcemia of any cause
- Ileal disease or resection (leads to increased oxalate absorption and urinary excretion)
- Renal tubular acidosis type I

Congenital and inherited causes:

- Familial hypercalciuria
- Renal tubular acidosis type I (distal)
- Medullary sponge kidney
- Cystinuria
- Primary hyperoxaluria

The majority of stones pass spontaneously within 48 hours. However, some stones may not. There are several factors which influence the ability to pass a stone. These include the size of the person, prior stone passage, prostate enlargement, pregnancy, and the size of the stone. A 4 mm stone has an 80% chance of passage while a 5 mm stone has a 20% chance. If a stone does not pass, certain procedures (usually by a urology specialist doctor) may be needed[66].

RISK FACTORS

Several risk factors are recognised to increase the potential of a susceptible individual to develop stones. These include:

- Anatomical anomalies in the kidneys and/or urinary tract - eg, horseshoe kidney, ureteral stricture.
- Family history of renal stones.
- Hypertension.
- Gout.
- Hyperparathyroidism.

- Immobilisation.
- Relative dehydration.
- Metabolic disorders which increase excretion of solutes - eg, chronic metabolic acidosis, hypercalciuria, hyperuricosuria.
- Deficiency of citrate in the urine.
- Cystinuria (an autosomal-recessive aminoaciduria).
- Drugs - eg, diuretics such as triamterene and calcium/vitamin D supplements.
- More common occurrence in hot climates.
- Increased risk of stones in higher socio-economic groups.
- Contamination - as demonstrated by a spate of melamine-contaminated infant milk formula[67].

EXAMINATION

- The patient with colic of any sort writhes around in agony. This is in contrast to the patient with peritoneal irritation who lies still.
- The patient is apyrexial in uncomplicated renal colic (pyrexia suggests infection and the body temperature is usually very high with pyelonephritis).
- Examination of the abdomen can sometimes reveal tenderness over the affected loin. Bowel sounds may be reduced. This is common with any severe pain.
- There may be severe pain in the testis but the testis should not be tender.
- Blood pressure may be low.
- Full and thorough abdominal examination is essential to check for other possible diagnoses - eg, acute appendicitis, ectopic pregnancy, aortic aneurysm[57.]

sDIFFERENTIAL DIAGNOSIS

This depends upon the position of the pain and the presence or absence of pyrexia and includes:

- Biliary colic.
- Pyelonephritis: very high temperature. Pain is unlikely to radiate to the groin.
- Acute pancreatitis

- Acute appendicitis
- Perforated peptic ulcer.
- Epididymo – orchitis or torsion of the testis: very tender testis.
- Sinister causes of back pain: usually tender over vertebrae.
- Dissection of an aortic aneurysm: the patient who presents with features of renal colic for the first time over the age of 60. This may be dissection of aortic aneurysm leading to ruptured aortic aneurysm.
- Drug addiction: there are reports of people with fictitious stories of renal colic, designed to obtain an injection of pethidine. These patients tend to be abusive when offered anything other than pethidine[68.]

INVESTIGATIONS

Basic analysis should include:

- Blood for FBC, CRP, renal function, electrolytes, calcium, phosphate and urate, creatinine.
- Midstream specimen of urine for microscopy (pyuria suggests infection), culture and sensitivities.
- Prothrombin time and international normalised ratio if intervention is planned.
- Stick testing of urine for red cells (suggestive of urolithiasis), white cells and nitrites (both suggestive of infection) and pH (pH above 7 suggests urea-splitting organisms such as *Proteus* spp. whilst a pH below 5 suggests uric acid stones).

- Intravenous pyelogram (IVP)[69] .
- Computed tomography (CT)
- Ultrasound scanning may be helpful to differentiate radio-opaque from radiolucent stones and in detecting evidence of obstruction.
- Plain X-rays of the kidney, ureter and bladder (KUB) are useful in watching the passage of radio-opaque stones (around 75% of stones are of calcium and so will be radio-opaque).

- The European Association of Urology's guidelines on urolithiasis recommend stone analysis for:

- All first-time stone formers.
- All patients with recurrent stones who are on pharmacological preventing therapy.
- Patients who have had early recurrence after complete stone clearance.
- Late recurrence after a long stone-free period (stone composition may change)[70].

COMPLICATIONS

The complications of calcium oxalate and hydroxyapatite renal calculi are acute and chronic pyelonephritis, hydronephrosis and obstructive nephropathy.

The stone fragments may obstruct the ureter. This occurs in 5-15% of cases. 8% of patients develop hypertension or exacerbation of pre- existing hypertension within 1 year. thirdly there is a risk of renal damage.

HYDRONEPHROSIS

In bilateral complete obstruction, patient present with anuria. When the obstruction is below the bladder the dominant symptoms are bladder distention. Unilateral hydronephrosis may remain completely silent for long periods of time. Enlargement of kidney is made out on physical examination. Sometimes the obstructing cause e.g. calculi can produce symptoms.

Early removal of the cause of obstruction can return the full function of the kidney. In long standing cases the changes become irreversible.

Two metabolic disorders need to be mentioned here which are associated with precipitation of the crystalline material causing obstruction to urine flow.

1.HYPERURICAEMIA

Uric acid stones are formed In 22% of patients with gout. The urate crystals get deposited in distal collecting tubules. *Collecting ducts as well in the interstitium, forming gouty tophus.* Uric acid crystal deposition takes place also following chemotherapy to the patients of leukemia and lymphoma. this is due to the breaking down of nucleic acid.

2.HYPERCALCAEMIA

Hyperparathyroidism, end stage kidney disease, vit. D intoxication, excessive calcium intake, osteolytic disease of bones, milk- alkali syndrome are some of the conditions giving rise to hypercalcaemia. This induces deposition of calcium in renal tubules called nephrocalcinosis. Sometimes, the deposition can also form renal stones[62].

Complete blockage of the urinary flow from a kidney decreases glomerular filtration rate (GFR) and, if it persists for more than 48 hours, may cause irreversible renal damage.

If ureteric stones cause symptoms after four weeks, there is a 20% risk of complications, including deterioration of renal function, sepsis and ureteric stricture.

Infection can be life-threatening.

Persisting obstruction predisposes to pyelonephritis[70].

PROGNOSIS

The prognosis will depend upon the underlying condition causing the renal stones. Calcium oxalate and hydroxyapatite stones per se rarely lead to renal failure. If at the time of diagnosis there is renal damage due to the calculi then this may cause renal dysfunction and hypertension. The main problem is recurrence unless the causative condition can be treated ; the recurrence rate is high, approaching 70% by 10 years after spontaneous passage or surgical removal of a calculus[71].

- Most symptomatic renal stones are small (less than 5 mm in diameter) and pass spontaneously.
- Stones less than 5 mm in diameter pass spontaneously in up to 80% of people.
- Stones between 5 mm and 10 mm in diameter pass spontaneously in about 50% of people.
- Stones larger than 1 cm in diameter usually require intervention (urgent intervention is required if complete obstruction or infection is present).
- Two thirds of stones that pass spontaneously will do so within four weeks of onset of symptoms.
- A stone that has not passed within 1-2 months is unlikely to pass spontaneously.
- The following features predispose to recurrent stone formation:
- First attack before 25 years of age.
- Single functioning kidney.
- A disease that predisposes to stone formation.
- Abnormalities of the renal tract.

PREVENTION

Recurrence of renal stones is common and therefore patients who have had a renal stone should be advised to adapt and adopt several lifestyle measures which will help to prevent or delay recurrence:

- Increase fluid intake to maintain urine output at 2-3 litres per day.
- Reduce salt intake.
- Reduce the amount of meat and animal protein eaten.
- Reduce oxalate intake (foods rich in oxalate include chocolate, rhubarb, nuts) and urate-rich foods (eg, certain fish).
- Drink regular cranberry juice: increases citrate excretion and reduces oxalate and phosphate excretion.

CALCIUM

- Maintain good calcium intake(calcium forms an insoluble salt with dietary oxalate, lowering oxalate absorption and excretion).
- Avoid calcium supplements separate from meals (increase calcium excretion without reducing oxalate excretion).

OXALATE

Depending on the composition of the stone, medication to prevent further stoneformation is sometimes given - eg, thiazide diuretics (for calcium stones),allopurinol (for uric acid stones) and calcium citrate (for oxalate stones)[57]

TRIAL DRUG

TRIAL DRUGS

வெடியுப்பு செந்தூரம்

INGREDIENTS:

வெடியுப்பு, வெற்றிலை

வெடியுப்பு

“மல்லாரு மட்டகுன்ம மாதருத ரக்கட்டி
கல்லா மதைப்புநீர்க் கட்டருக- லெல்லாமே
கம்பிகம்பி யென்றுங் கருவுண்டா மங்கிநின்ற
கம்பிகம்பி யென்றுரைக்குங் கால்.”[72]

வெற்றிலை

ஐயம் அறுங்காண் அதன்சாரங் கொண்டக்காற்
பையச் சயித்தியம்போம் பைந்தொடியே !- மெய்யின்
கடியின் குணம் போகுங் காரவெற்றி லைக்குப்
படியுமுத் தோடமிதைப் பார்.[72]

ACTIONS OF TRIAL DRUGS

S.No	Drugs	Botanical name	Actions
1	வெடியுப்பு	Potassium nitrate	குளிர்ச்சியுண்டாக்கி, சிறுநீர்பெருக்கி, வியர்வைபெருக்கி.[72]
2	வெற்றிலை	Piper betle	வெப்பகற்றி, பால்பெருக்கி, பசித்தூண்டி,துவர்ப்பி, அழுகலகற்றி,அகட்டு வாய்வகற்றி,உமிழ்நீர்ப் பெருக்கி, காமம்பெருக்கி, வெப்பமுண்டாக்கி.73

STANDARD OPERATIVE PROCEDURE

Take 175gms of Potash nitras a clay pot, heat it over fire and the salt will melt. By this time add Betle leaf juice about 168ml, stir it un till the juice gets immerge with the salt. Pour this over a wooden plate or a clean Kalvam.

Then transfer it into a pot and heat by adding Betel leaf juice till the juice immerge with the ingredient and become semi solid state. Repeat the same procedure for 3 times.

Now the color will turn into red. Otherwise repeat this till the color changes in to red let it cool and dry then bottle it up.

DURATION: 48 days(1mandalam)

DOSE: 130 mg to 260 mg bd.

ADJUVANT: Mullangi kizhangu Chaaru[74]

INGREDIENTS OF TRIAL DRUGS

வெற்றிலை

BEFORE PURIFICATION OF VEDIYUPPU

AFTER PURIFICATION OF VEDIYUPPU

TRIAL MEDICINE

MATERIALS AND METHODS

MATERIALS AND METHODS

PROTOCOL

Study Design

An open pilot study on kalladaippu was carried out in the post graduate department of maruthuvam in Govt.Siddha Medical College attached to Arignar Anna Hospital of Indian Medicine, Chennai – 106 during the period of 2014 – 2016.

The study was approved by **Instititional Ethics Committee (IEC)** and the approval number is **GSMC-CH-ME-3/001/2014.**

Sample size

The study is conducted in 40 selected kalladaippu patients of both genders between age groups of 18 to 60 years.

Selection Criteria

The patients having following parameters are selected for the study.

- Pain in the flank
- Burning Micturition
- Oliguria
- Dysuria
- Nausea
- Vomiting
- Haematuria
- Fever
- History of Urolithiasis (with USG-Whole Abdomen reports)

Exclusion Criteria

- Age group less than 18
- Stag horn calculus
- Pyonephrosis
- Calculi associated with elevated serum creatinine level
- Calculi in pregnancy

Proforma

The case sheet proforma for kalladaippu was prepared based on Siddha diagnostic methodology with necessary modern techniques.

History taking

For better treatments and results a detailed clinical history was taken regarding present illness, past illness, family history, menstrual history, occupational history, socio economic status, residential area, etc.,

Investigation

All patients were screened by the following investigations. This was carried out regularly before and after treatment.

- **Blood for biochemical examination**

  The blood was tested for sugar, urea, serum creatinine to know the renal function and its excretion.

- **Urine Examination**

  Albumin, Sugar, Deposits.

- **Ultra sonogram**

  Ultra sonogram of complete abdomen including KUB was done in cases to know the location, size and number of calculi.

Drug and dose schedule

Vediyuppu chendooram– 130 mg to 260 mg, bd after food with radish juice for 48 days

RESULTS

AND

OBSERVATION

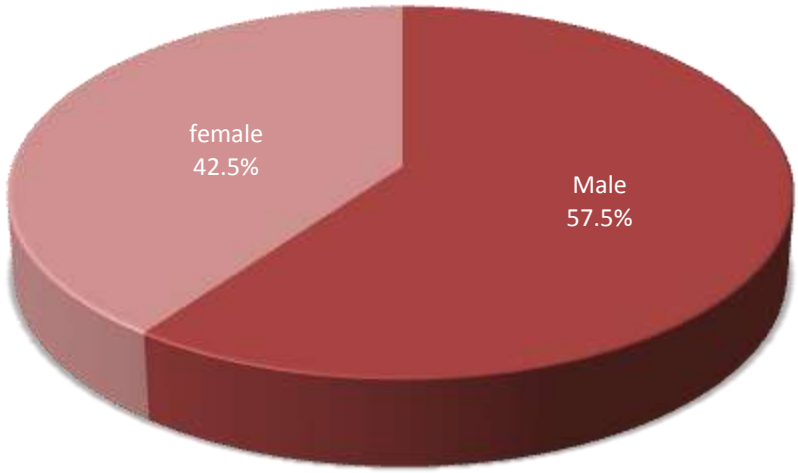
RESULTS AND OBSERVATION

40 cases having kalladaippu were selected and treated in OPD of PG maruthuvam department attached to AAGHIM, Chennai – 106 during the year 2014 – 2016. The result and observation during that clinical study are as follows.

- Gender distribution
- Age distribution
- Occupation
- Socio- economic status
- Dietary habits
- Seasonal occurence
- Distribution of thinai
- Distribution of mukkutram – vatham
- Distribution of mukkutram – pitham
- Distribution of mukkutram – kabham
- Ezhu udal thathukkal
- En vagai thervugal
- Naadi
- Neikuri
- Clinical features
- Clinical prognosis
- Distribution of calculi based on location
- Grading of results

GENDER DISTRIBUTION

S.No	GENDER	NUMBER OF CASES	PERCENTAGE (%)
1	Male	23	57.5%
2	Female	17	42.5%



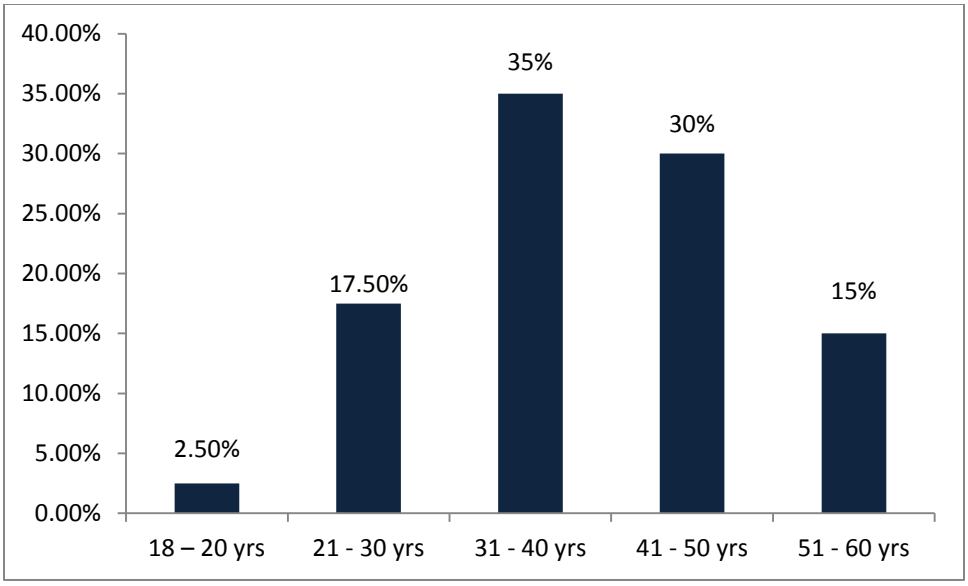
INFERENCE

About 57.5% were males and 42.5% were females

Literature: according to literature males are more prone to renal calculi

AGE DISTRIBUTION

S.No	AGE IN YEARS	NUMBER OF CASES	PERCENTAGE (%)
1	18 – 20 yrs	1	2.5%
2	21 - 30 yrs	7	17.5%
3	31 - 40 yrs	14	35%
4	41 - 50 yrs	12	30%
5	51 - 60 yrs	6	15%

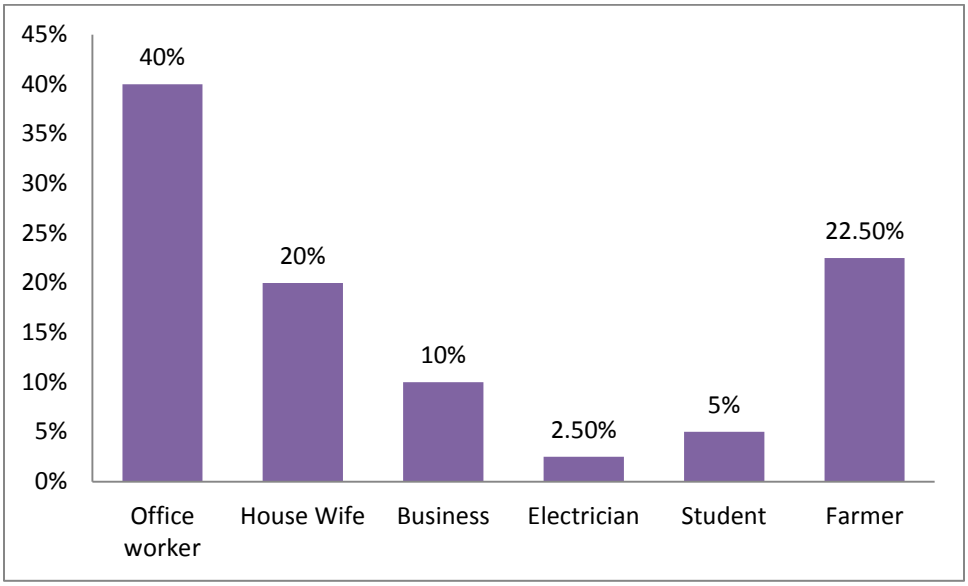


INFERENCE

Majority of the case that is 35% were in the 3rd decade, 30% were in the 4th decade, 17.5% were in the 2nd decade, 15% were in the 5th decade, 2.5%were in 1st decade.

OCCUPATION

S.No	OCCUPATION	NUMBER OF CASES	PERCENTAGE (%)
1	Office worker	16	40%
2	House Wife	8	20%
3	Business	4	10%
4	Electrician	1	2.5%
5	Student	2	5%
6	Farmer	9	22.5%

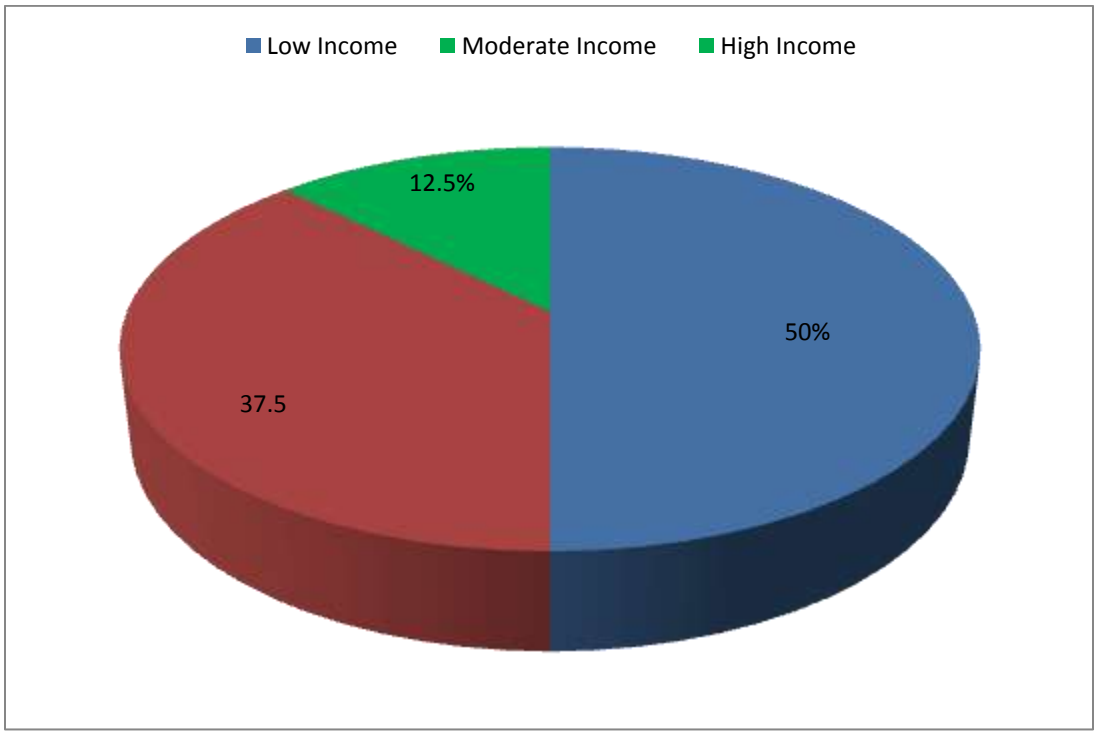


INFERENCE

0ut of 40 patients (100%), 40% were office worker, 20% were house wife, 10% were business, 2.5% were electrician, 5% were student, 22.5% were farmer.

SOCIO – ECONOMIC STATUS

S.No	SOCIO – ECONOMIC STATUS	NUMBER OF CASES	PERCENTAGE (%)
1	Low Income (below 25,000 per annum)	20	50%
2	Moderate Income (25,000 – 50,000 per annum)	15	37.5%
3	High Income (Above 50,000 per annum)	5	12.5%

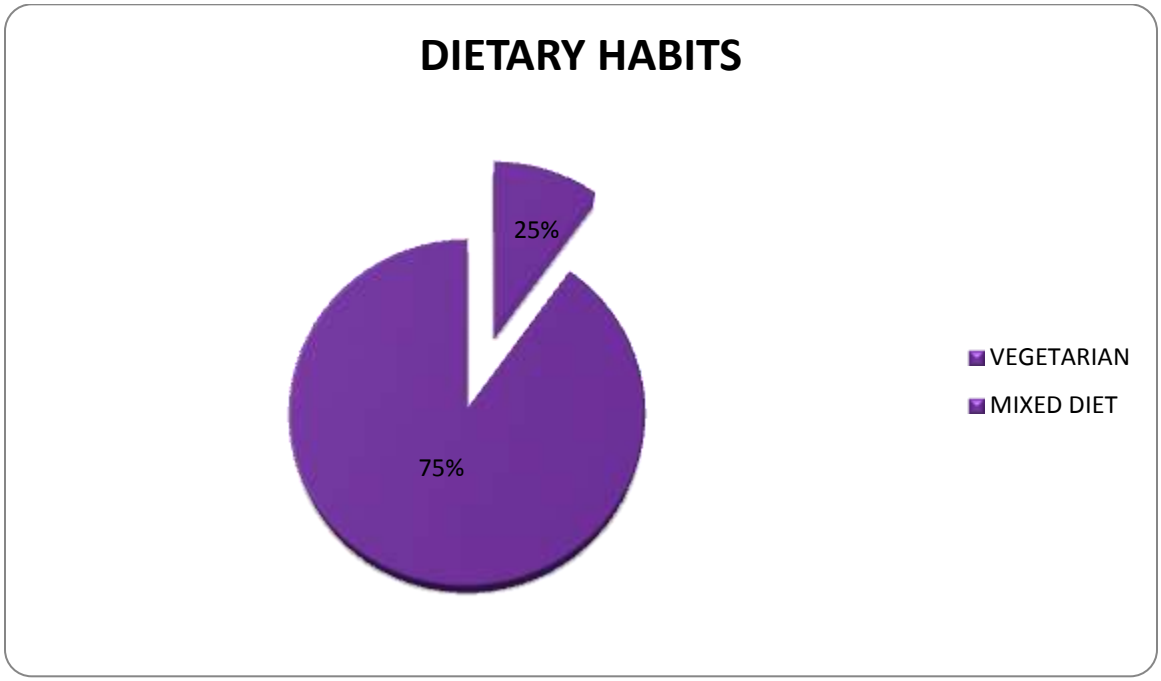


INFERENCE:

Among 40 cases 50% comes under low economic status, 37.5% of them under moderate status and 12.5% of them under high income status.

DIETARY HABITS

S.No	DIET	NUMBER OF CASES	PERCENTAGE (%)
1	Vegetarian	10	25%
2	Mixed diet	30	75%

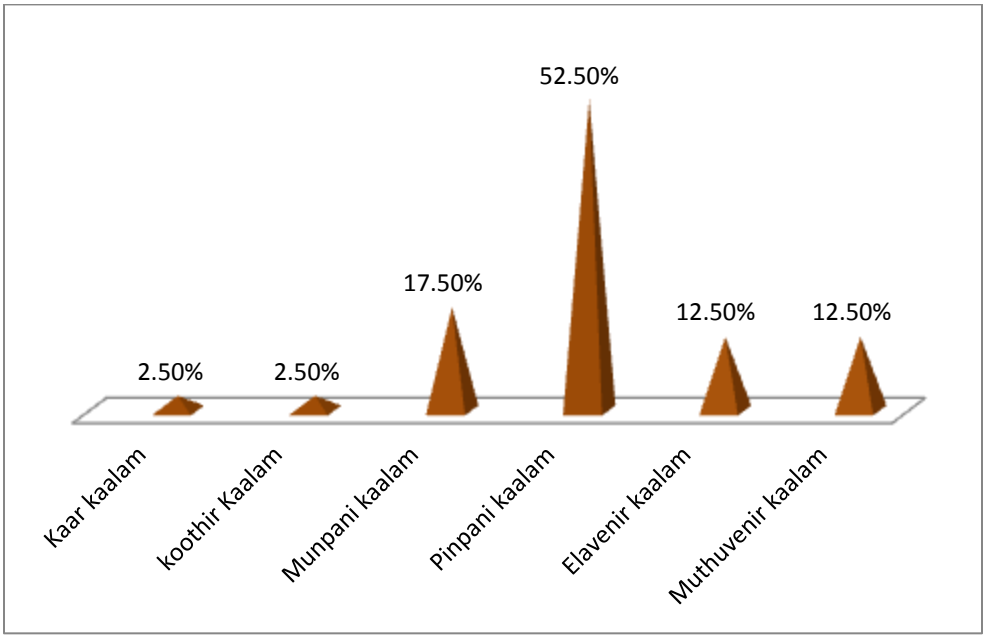


INFERENCE

Among 40 patients, 10 patients (25%) were taking vegetarian food and 30 patients (75%) were taking mixed diet.

SEASONAL OCCURENCE

S.No	KAALAM (Season)	NUMBER OF CASES	PERCENTAGE (%)
1	Kaar kaalam (ஆவணி, புரட்டாசி)	1	2.5%
2	koothir Kaalam (ஐப்பசி, கார்த்திகை)	1	2.5%
3	Munpani kaalam (மார்கழி, தை)	7	17.5%
4	Pinpani kaalam (மாசி, பங்குனி)	21	52.5%
5	Elavenir kaalam (சித்திரை, வைகாசி)	5	12.5%
6	Muthuvenir kaalam (ஆனி, ஆடி)	5	12.5%

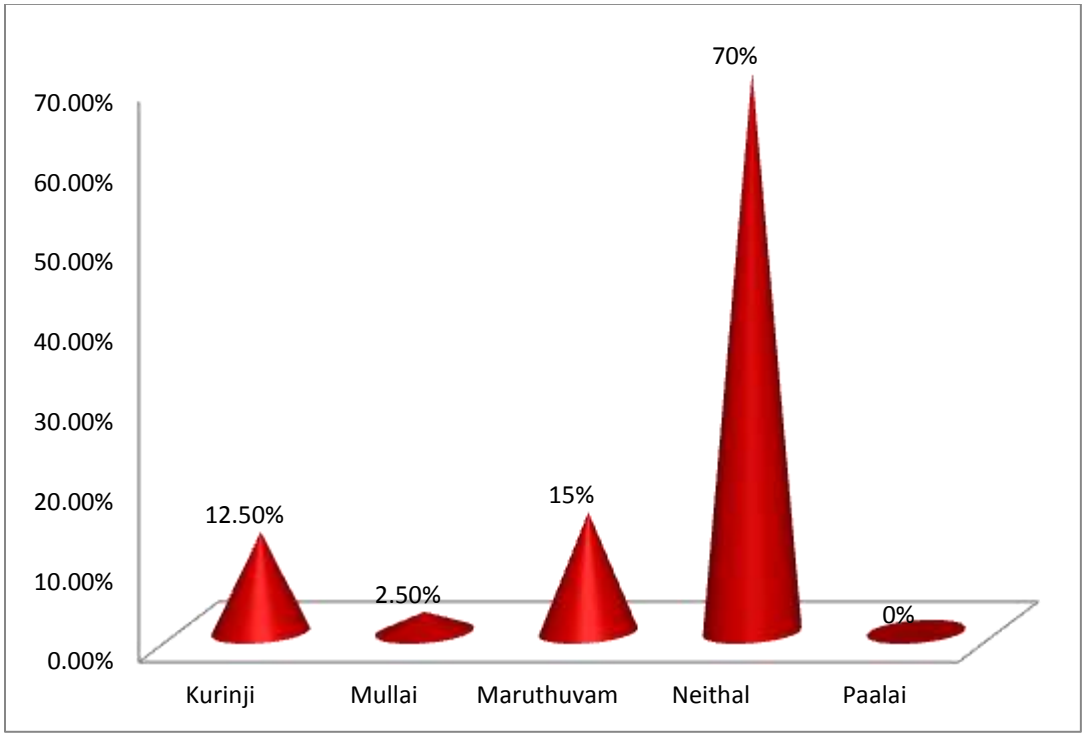


INFERENCE

According to paruvakaalam highest incident of 21 cases (52.5%) were noted in pinpani kaalam , 7 cases (17.5%) were noted in munpani kaalam, 5 cases (12.5%) were noted in elavenir kaalam, 5 cases (12.5%) were noted in muthuvenir kaalam, 1 case (2.5%) were noted in karkaalam And 1 case(2.5%) were noted in koothir kaalam.

DISTRIBUTION OF THINAI

S.No	THINAI	NUMBER OF CASES	PERCENTAGE (%)
1	Kurinji	5	12.5%
2	Mullai	1	2.5%
3	Marutham	6	15%
4	Neithal	28	70%
5	Paalai	0	0%

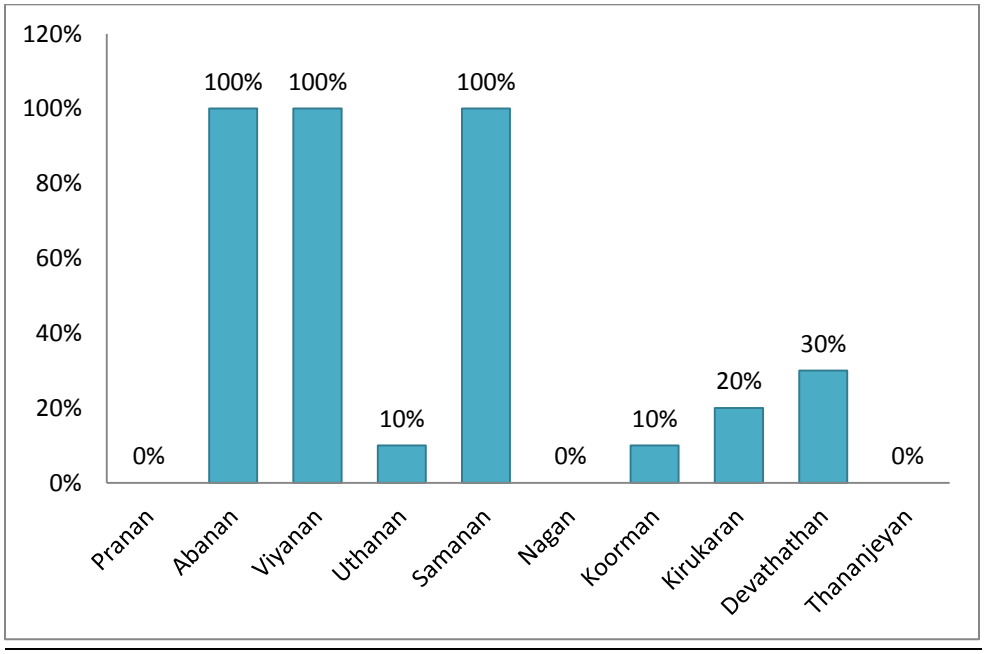


INFERENCE

According to thinai the highest distribution 70% was noted in neithal, 15% in marutham, 12.5% in kurinji, 2.5% in mullai.

DISTRIBUTION OF MUKKUTRAM – VATHAM

S.No	VATHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Pranan	0	0%
2	Abanan	40	100%
3	Viyanan	40	100%
4	Uthanan	4	10%
5	Samanan	40	100%
6	Nagan	0	0%
7	Koorman	4	10%
8	Kirukaran	8	20%
9	Devathathan	12	30%
10	Thananjeyan	0	0%

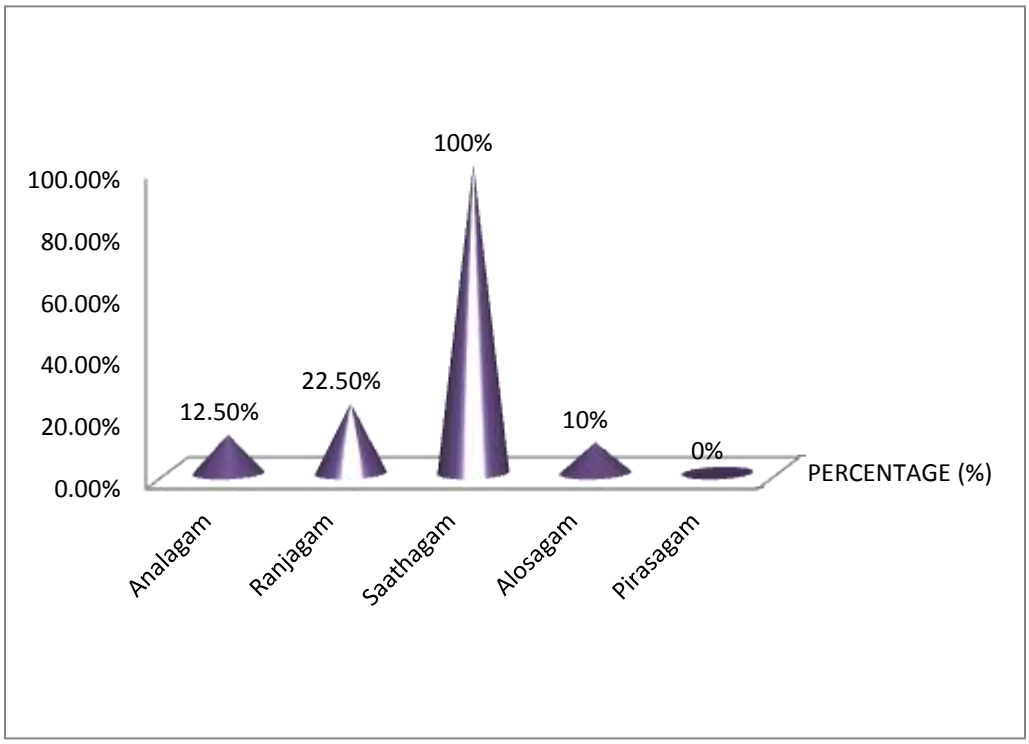


INFERENCE

Out of 40 patients Abanan was affected in 40 patients (100%), Viyanan was affected in 40 pateints (100%), Uthanan was affected in 4 patients (10%), Samanan was affected in 40 patients (100%), Koorman was affected in 4 patients (10%) and Kirukaran was affected in 8 patients (20**%),**Devathathan was affected in 12 patients(30%).

DISTRIBUTION OF MUKKUTRAM – PITHAM

S.No	PITHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Analagam	5	12.5%
2	Ranjagam	10	22.5%
3	Saathagam	40	100%
4	Alosagam	4	10%
5	Pirasagam	0	0%

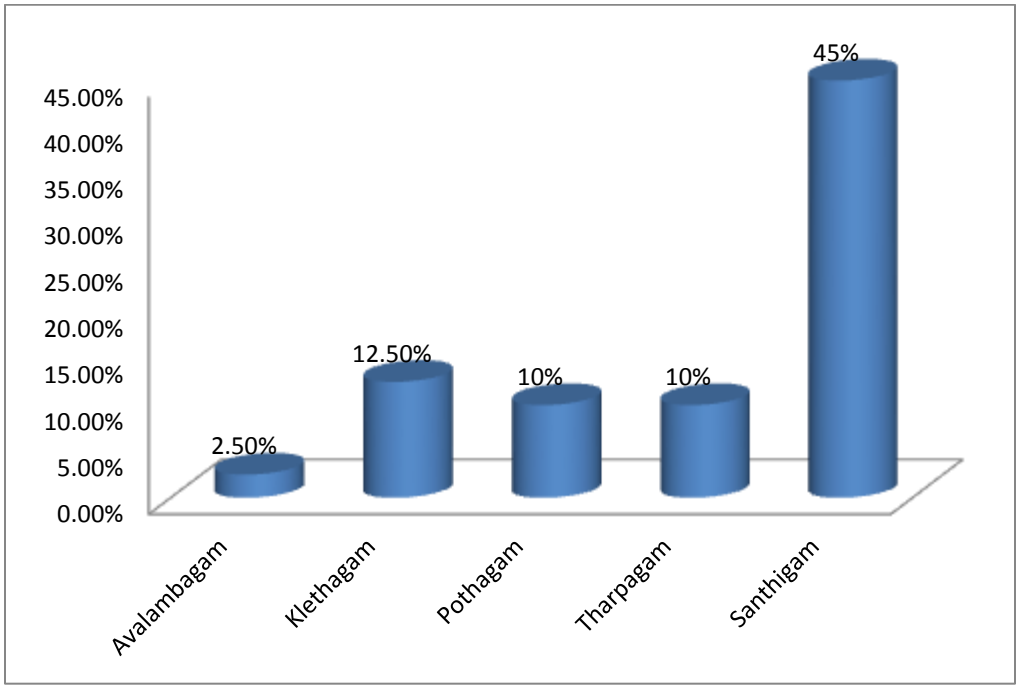


INFERENCE

Out of 40 patients Analagam was affected in 5 patients (12.5%), Ranjagam was affected in 10 patients (22.5%), Sathagam was affected in 40 patients (100%), Aalosagam was affected in 4 patients (10%).

DISTRIBUTION OF MUKKUTRAM – KABHAM

S.No	KABHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Avalambagam	1	2.5%
2	Klethagam	5	12.5%
3	Pothagam	4	10%
4	Tharpagam	4	10%
5	Santhigam	18	45%

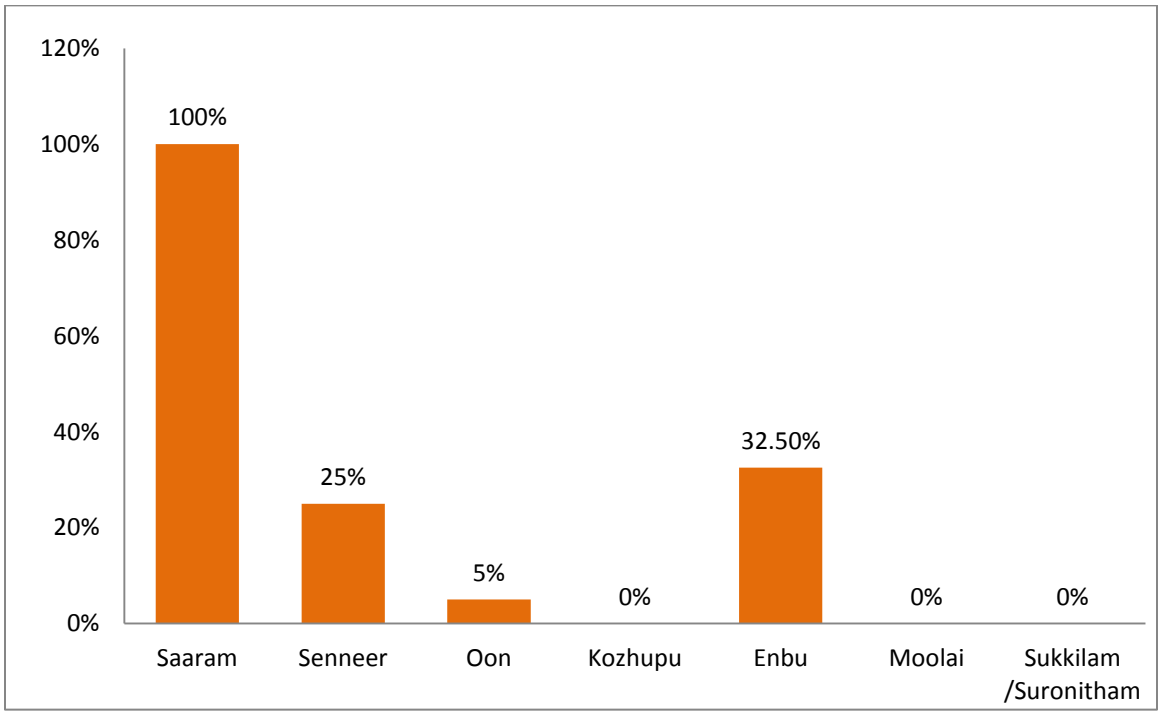


INFERENCE

Out of 40 patients, Avalambagam was affected in one patient(2.5%), Kiledhagam was affected in 5 patients (12.5%), Pothagam was affected in 4patients(10%), Tharpagam was affected in 4 patients(10%), Santhigam was affected in18 patients (45%).

EZHU UDAL THATHUKAL

S.No	EZHU UDAL THATHUKAL	NUMBER OF CASES	PERCENTAGE (%)
1	Saaram	40	100%
2	Senneer	10	25%
3	Oon	2	5%
4	Kozhupu	0	0%
5	Enbu	13	32.5%
6	Moolai	0	0%
7	Sukkilam /Suronitham	0	0%

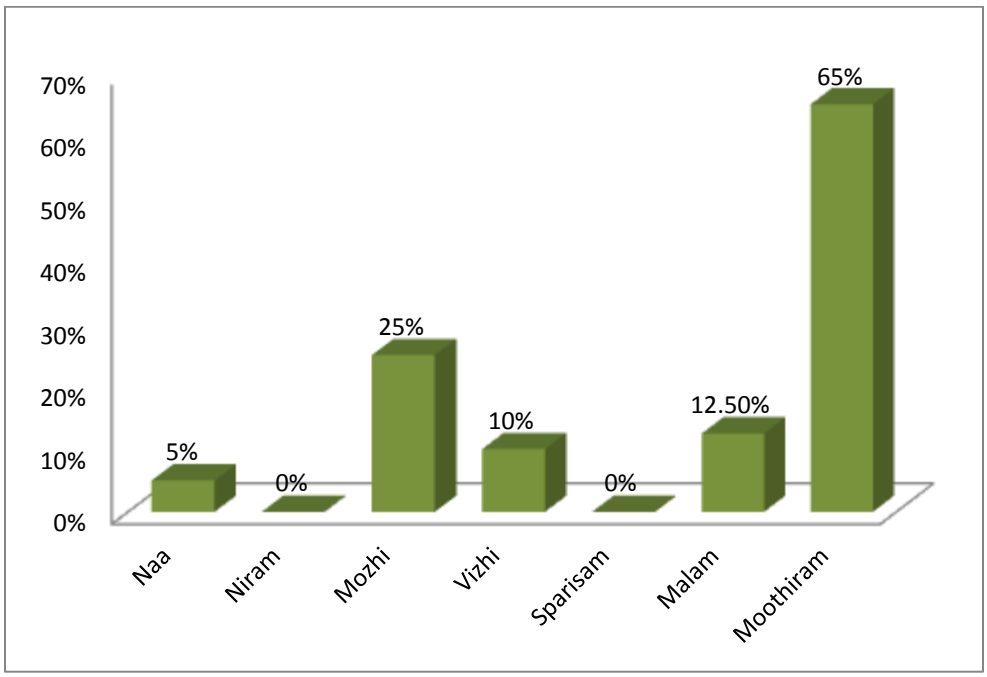


INFERENCE

Out of 40 patients, Saaram was affected in 40 patients (100%), Senneer was affected in 10 patients (25%), Oon was affected in 2 patients(5%), Enbu was affected in 13 patients (32..5%).

EN VAGAI THERVUGAL

S.No	EN VAGAI THERVUGAL	NUMBER OF CASES	PERCENTAGE (%)
1	Naa	2	5%
2	Niram	0	0%
3	Mozhi	10	25%
4	Vizhi	4	10%
5	Sparisam	0	0%
6	Malam	5	12.5%
7	Moothiram	26	65%

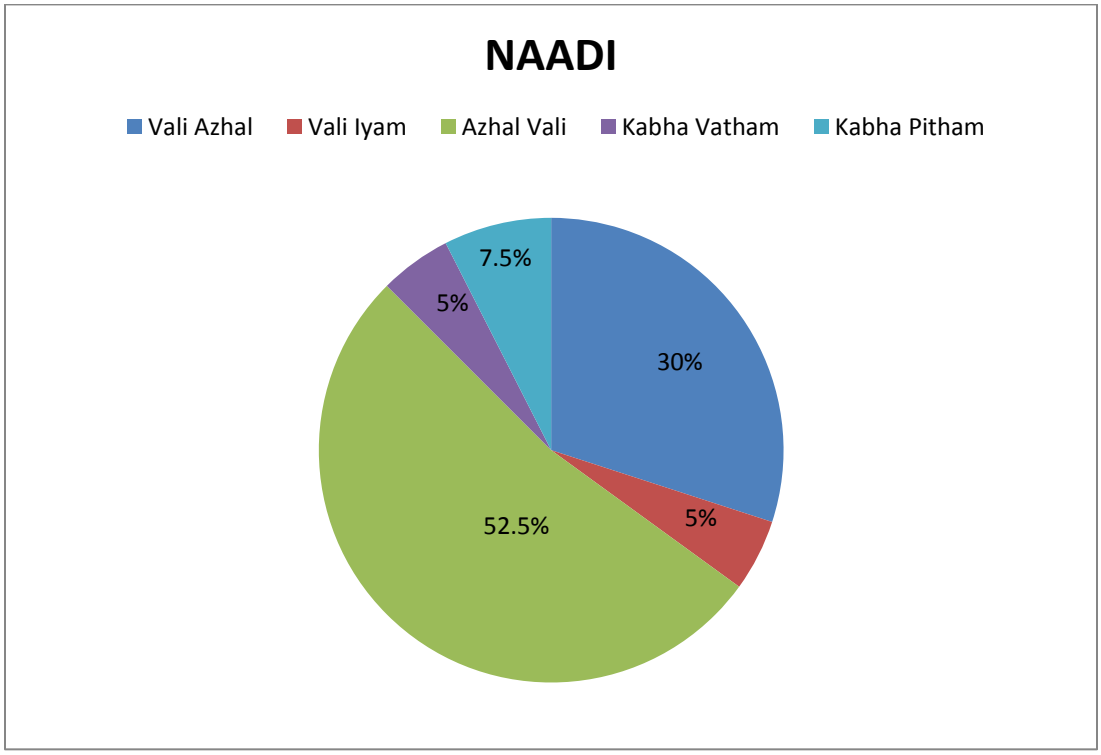


INFERENCE

In Envagai thervu,Naa was affected in 2 patients(5%), Mozhi was affected in 10 patients(25%), Vizhi was affected in 4 patients (10%), Malam was affected in 5 patient (12.5%) and Moothiram was affected in 26 patients (65%).

NAADI

S.No	NAADI	NUMBER OF CASES	PERCENTAGE (%)
1	Vali Azhal	12	30%
2	Vali Iyam	2	5%
3	Azhal Vali	21	52.5%
4	Kabha Vatham	2	5%
5	Kabha Pitham	3	7.5%

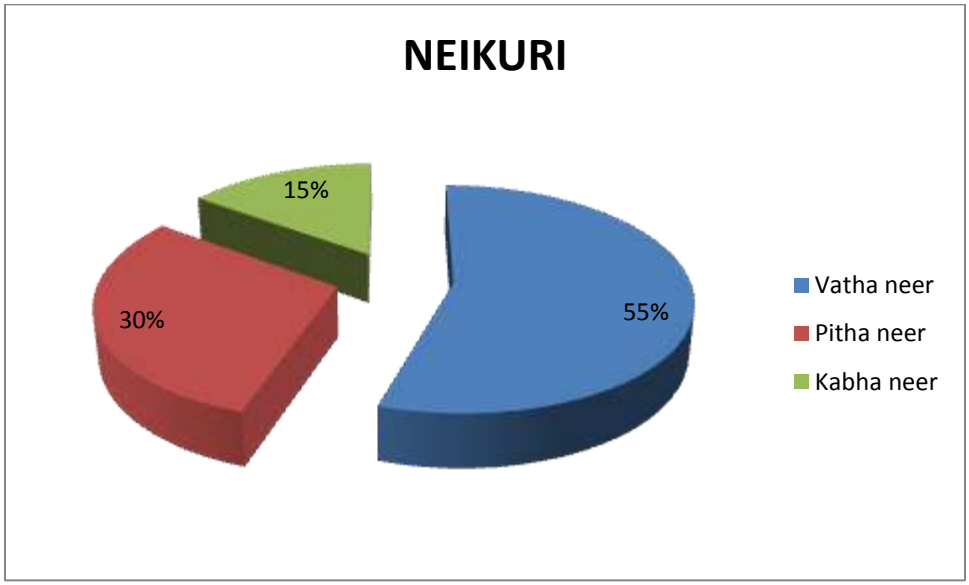


INFERENCE

12 patients (30%) had Vali azhal naadi, 3 patients (5%) had vali Iyam naadi, 21 patients (52.5%) had Azhal vali naadi,2 patients (5%) had kabha vatham, and 3 patients (7.5%) had Kabha pitha naadi.

NEIKURI

S.No	THATHU	NEIKURI	NUMBER OF CASES	PERCENTAGE (%)
1	Vatha neer	Spread like snake	22	55%
2	Pitha neer	Spread like ring	12	30%
3	Kabha neer	Spread like pearl	6	15%

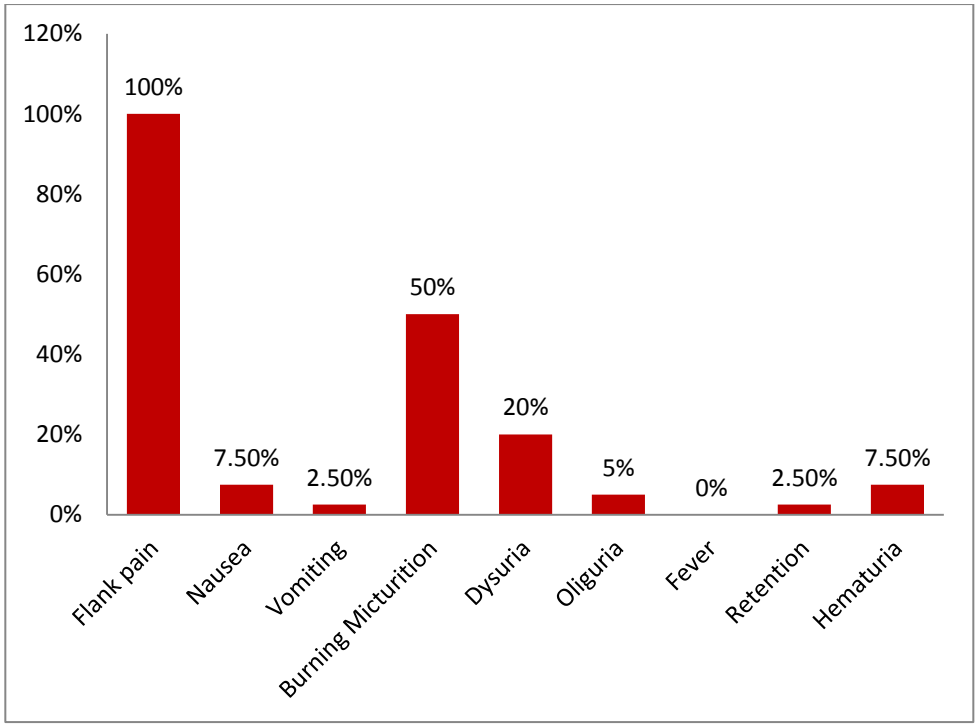


INFERENCE

22 patients (55%) had vatha neer, 12 patients (30%) had Pitha neer, and 6 patients (15%) had Kabha neer.

CLINICAL FEATURES

S.No	SIGNS & SYMPTOMS	NUMBER OF CASES	PERCENTAGE (%)
1	Flank pain	40	100%
2	Nausea	3	7.5%
3	Vomiting	1	2.5%
4	Burning Micturition	20	50%
5	Dysuria	8	20%
6	Oliguria	2	5%
7	Fever	0	0%
8	Retention	1	2.5%
9	Hematuria	3	7.5%

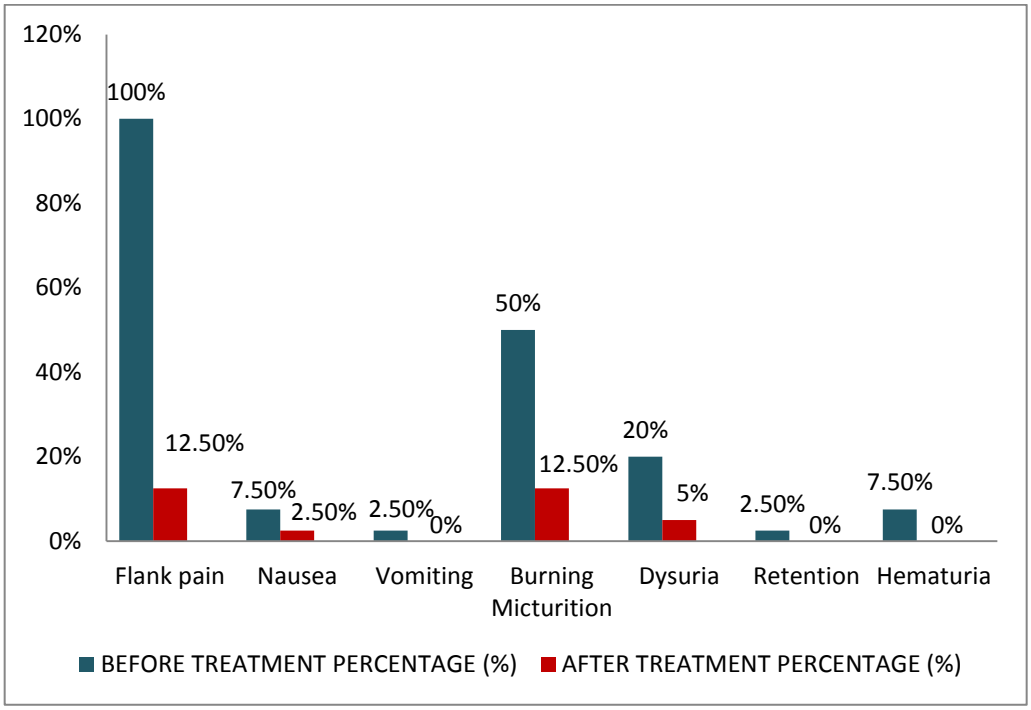


INFERENCE

Out of 40 patients, 40 patients (100%) had Flank pain, 3 patients (7.5%) had Nausea, 1 patient (2.5%) had vomiting,20 patients (50%) had Burning Micturition, 8patients (20%) had Dysuria, 2 patients (5%) had oliguria, 1 patient(2.5%) had urine retion, and 3 patients (7.5%) had Hematuria

CLINICAL PROGNOSIS

S.No	SIGNS& SYMPTOMS	BEFORE TREATMENT		AFTER TREATMENT	
		NO.OF CASES	PERCENTAGE (%)	NO.OF CASES	PERCENTAGE (%)
1.	Flank pain	40	100%	9	12.5%
2	Nausea	3	7.5%	1	2.5%
3	Vomiting	1	2.5%	0	0%
4	Burning Micturition	20	50%	5	12.5%
5	Dysuria	8	20%	2	5%
6	Retention	1	2.5%	0	0%
7	Hematuria	3	7.5%	0	0%

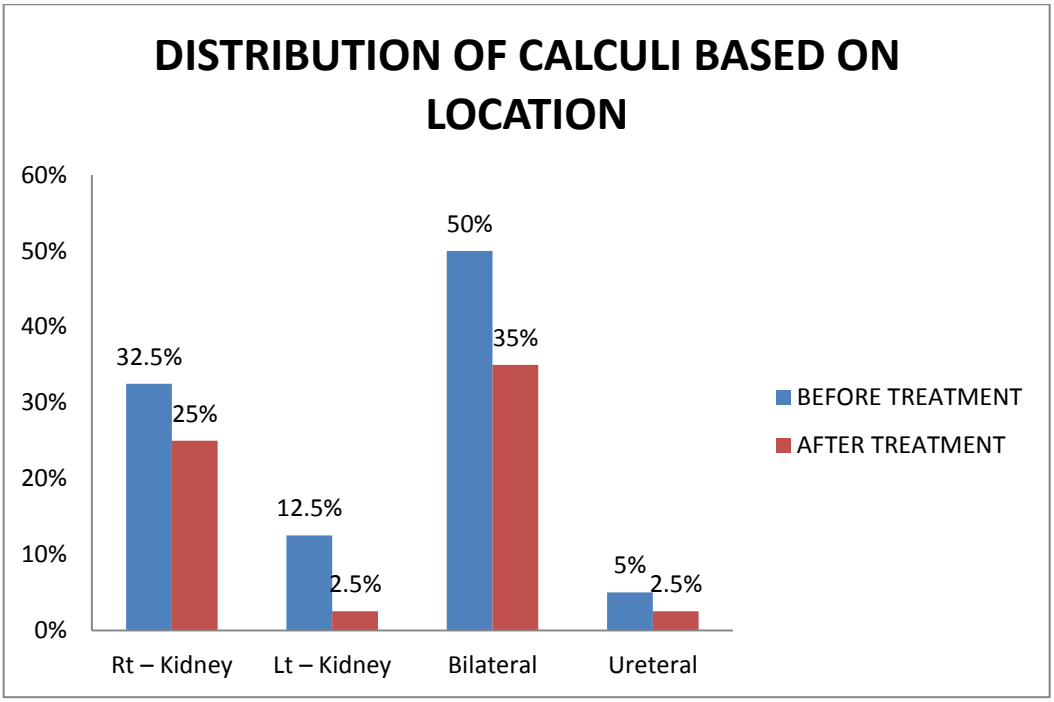


INFERENCE

After treatment Flank pain present in 5 patients (12.5%), nausea present in one patient (2.5%), Burning Micturition present in 5patient (12.5%), Dysuria present in 2 patients (5%).

DISTRIBUTION OF CALCULI BASED ON LOCATION

SIDE	BEFORE TREATMENT		AFTER TREATMENT	
	NO.OF	PERCENTAGE	NO.OF	PERCENTAGE
Rt – Kidney	13	32.5%	10	25%
Lt – Kidney	5	12.5%	1	2.5%
Bilateral	20	50%	14	35%
Ureteral	2	5%	1	2.5%



DISTRIBUTION OF CALCULI BASED ON LOCATION

INFERENCE

Before treatment: Out of 40 patients, 20 patients (50%) were having bilateral renal calculi, 13 patients (32.5%) were having right renal calculi, and 5 patients (12.5%) were having left renal calculi, 2 patients were having ureteric calculi.

After treatment:

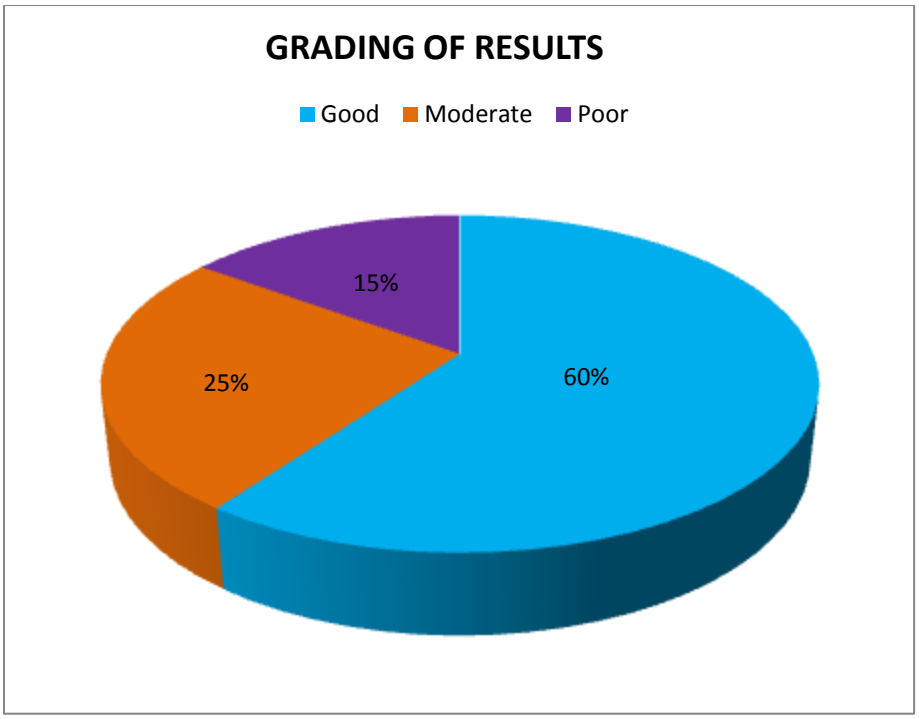
Out of 40 patients, 14 patients (35%) were having bilateral renal calculi, 10 patients (25%) having right renal calculi and 1 patient (2.5%) having left renal calculi, 1 patient (2.5%) having ureteric calculi.

GRADING OF RESULTS

S.NO	NO OF CASES	BEFORE TREATMENT	AFTER TEATMENT	
			Expelsion of calculi	Size reducing of calculi
1	6	4 mm- 5mm	4patients	3mm, 3.5mm
2	8	5 mm- 6mm	5 patients	3mm,4mm,5mm.
3	3	6 mm-7mm	1 patient.	3mm, 5mm.
4	1	7 mm-8mm		3.2mm
5	5	8 mm-9mm		5mm,7mm,3.1mm, 7mm,8mm.
6	1	10 mm	1 patient	
7	1	11 mm		4mm
8	15	Multiple stones	3 patients	12 patients has size reducing of calculi.

GRADING OF RESULTS

S.No	GRADING	NUMBER OF CASES	PERCENTAGE (%)
1	Good	24	60%
2	Moderate	10	25%
	Poor	6	15%



INFERENCE

Out of 40 patients, 24 cases (60%) shows good result, 10 cases (25%) shows moderate result, 6 cases (15%) shows poor result.

LIST OF PATIENTS

S.no	Op. no	Patient's name	Age/Sex	Occupation	Date of Medicine
1	5893	Mr. Karnan	37/M	Coolie	02-02-15
2	6705	Mr. Udhaya prakash	27/M	Engineer	06-02-15
3	8669	Mr. Ragavan	44/M	Clerk	18-02-15
4	271	Mr.Rjesh	25/M	Coolie	27-02-15
5	9055	Mrs.Anjammal	56/F	Housewife	05-03-15
6	3300	Mr.Vengateshan	31/M	Electrician	18-03-15
7	1546	Mrs.Lalitha	28/M	housewife	15-04-15
8	1723	Mrs.Selvi	49/F	Tailor	16-04-15
9	2846	Mrs. Malliga	44/F	House wife	20-04-15
10	2805	Mr.Dinesh kumar	20/M	Student	20-04-15
11	4176	Mr. Vasudevan	59/M	Teacher	24-04-15
12	9182	Mr.Mogamed	26/M	Clerk	12-05-15
13	2623	Mrs. Indira	31/F	Housewife	29-06-15
14	3288	Mrs.Amirthavalli	34/F	Housewife	01-07-15
15	3787	Mrs. Kanagavalli	52/F	Doctor	02-07-15
16	7792	Mr.Rajen	32/M	Coolie	16-07-15
17	5469	Mr.Sigamani	43/M	Mechanic	12-08-15
18	9907	Mr. Amulraj	43/M	Painter	28-08-15
19	9126	Mrs.Jeyabharathi	35/F	Teacher	03-11-15
20	1651	Mrs.Sundari	30/F	IT Profession	06-01-16
21	2186	Mr.Jeyachandran	46/M	Farmer	16-01-16
22	5220	Mrs.Lalitha	30/F	Teacher	20-01-16
23	6456	Mrs. Bavya	35/F	House wife	24-01-16
24	6455	Mrs.Anitha	33/F	IT profession	24-01-16
25	6880	Mr.Soundarrajen	31/M	Bank officer	25-01-16
26	7582	Mrs. Rajeshwari	36/F	House wife	27-01-16

27	9414	Mr. Rajaram	33/M	Teacher	1-02-16
28	9711	Mr.Kumar	37/M	Coolie	02-02-16
29	1727	Mr.Babu suresh	49/M	Office work	08-02-16
30	1935	Mrs.Umamaheshwari	30/F	Housewife	08-02-16
31	4590	Mrs.Shanthi	52/F	Tailour	16-02-16
32	5736	Mrs.Geetha	52/F	Housewife	18-02-16
33	6226	Mr. Murali	44/M	Farmer	20-02-16
34	6912	Mr.Ranganathan	60/M	Watchmen	23-02-16
35	7167	Mrs.Rajeshwari	36/F	Business	23-02-16
36	7498	Mr.Deena dhayalan	38/M	Clerk	24-02-16
37	7732	Mr. Rajasekar	45/M	Auto driver	24-02-16
38	9213	Mrs.Jeyanthi	42/F	Tailour	29-02-16
39	4885	Mr.Viveganathar	45/M	Driver	17-03-16
40	6097	Mr Shanmugasundaram	46/M	Farmer	21-03-16

S.No	Name of the patient	Age/Sex	Date of Treatment Started	Duration of medicine taken	Size of stone BT	Prognosis	Remarks
1	Mr. Karnan	37/M	02-02-15	7- Weeks	LT ureter-11mm	LT- 4mm	Symptoms reduced
2	Mr. Udhaya prakash	27/M	06-02-15	7- Weeks	Bilateral 4.5 mm	USG Normal	Completed
3	Mr. Ragavan	44/M	18-02-15	7- Weeks	Bilateral RT -3mm, LT-3mm	USG Normal	Symptoms reduced
4	Mr.Rjesh	25/M	27-02-15	7- Weeks	RT 7.3mm	RT 3.2mm	Symptoms reduced
5	Mrs.Anjammal	56/F	05-03-15	7- Weeks	RT (5 mm)	USG Normal	Symptoms reduced
6	Mr.Vengateshan	31/M	18-03-15	5- Weeks	Bilateral Muliple calculi(5mm)	RT 5mm, LT 3 (4mm,3mm)	Advice to continue medicine
7	Mrs.Lalitha	28/M	15-04-15	7- Weeks	RT 5mm	USG Normal	Symptoms relieved
8	Mrs.Selvi	49/F	16-04-15	7- Weeks	RT (10mm, 9mm)	RT 3mm	Symptoms relieved
9	Mrs. Malliga	44/F	20-04-15	7- Weeks	RT (8mm)	RT (5mm)	Symptoms reduced
10	Mr.Dinesh kumar	20/M	20-04-15	7- Weeks	Bilateral -RT2(6mm) LT multiple(5mm)	RT 2(4mm), LT 3 (4.5mm, 4mm, 3mm)	Symptoms reduced
11	Mr. Vasudevan	59/M	24-04-15	7- Weeks	Bilateral LT 9mm, 1.1cmRT 5mm	LT 9mm, RT 3mm	Symptoms reduced
12	Mr.Mogamed	26/M	12-05-15	7- Weeks	LT 6mm	5mm	Symptoms relieved
13	Mrs. Indira	31/F	29-06-15	7- Weeks	Bilateral RT 8mm, LT 8mm	RT 3mm, LT 3mm	Symptoms reduced
14	Mrs.Amirthavalli	34/F	01-07-15	7- Weeks	RT (4mm)	RT(3.5mm)	Symptoms reduced
15	Mrs. Kanagavalli	52/F	02-07-15	7- Weeks	LT ureter -5mm	LT-4mm	Symptoms reduced
16	Mr.Rajen	32/M	16-07-15	7- Weeks	LT-6mm	Stone	Symptoms relieved

						expelled	
17	Mr.Sigamani	43/M	12-08-15	7- Weeks	RT 5 mm	USG Normal	Symptoms reduced
18	Mr. Amulraj	43/M	28-08-15	7- Weeks	LT- 5mm	LT 3mm .	Symptoms relieved
19	Mrs.Jeyabharathi	35/F	03-11-15	7- Weeks	LT - 9mm,3mm	LT- 3mm	Symptoms reduced
20	Mrs.Sundari	30/F	06-01-16	7- Weeks	RT (4.5mm)	USG Normal	Symptoms relieved
21	Mr.Jeyachandran	46/M	16-01-16	7- Weeks	Bilateral RT-4.1mm , LT-4.2mm	RT 4mm.	Symptoms relieved
22	Mrs.Lalitha	30/F	20-01-16	7- Weeks	RT 10mm	Stone expelled	Completed
23	Mrs. Bavya	35/F	24-01-1R6	7- Weeks	RT (8.5mm)	RT (7mm)	Symptoms relieved
24	Mrs.Anitha	33/F	24-01-16	7- Weeks	RT 8mm	RT 8mm	Symptoms relieved
25	Mr.Soundarrajen	31/M	25-01-16	7- Weeks	Bilateral RT 4(5mm), LT 2(4, 3mm)	RT 3 mm, 4mm	Symptoms reduced
26	Mrs. Rajeshwari	36/F	27-01-16	7- Weeks	LT 5mm	USG Normal	Symptoms reduced
27	Mr. Rajaram	33/M	1-02-16	7- Weeks	RT (4.6mm)	RT (3mm)	Symptoms relieved
28	Mr.Kumar	37/M	02-02-16	7- Weeks	Bilateral RT 3 (6mm,3mm,3mm) LT multiple(5mm)	RT 3 (3mm), LT 2 (3.5mm)	Symptoms reduced
29	Mr.Babu suresh	49/M	08-02-16	7- Weeks	RT 6 mm	RT (3mm)	Symptoms reduced
30	Mrs.Umamaheshwari	30/F	08-02-16	7- Weeks	Bilateral RT (8.5mm), LT (8.5mm)	RT (3mm), LT (3.5mm)	Symptoms reduced
31	Mrs.Shanthi	52/F	16-02-16	7- Weeks	LT (8.7mm)	7mm	Symptoms relieved(
32	Mrs.Geetha	52/F	18-02-16	7- Weeks	RT (4.9mm),	USG Normal	Symptoms relieved
33	Mr. Murali	44/M	20-02-16	7- Weeks	Bilateral RT(4mm), LT (7mm)	USG Normal	Symptoms relieved

34	Mr.Ranganathan	60/M	23-02-16	7- Weeks	RT 2 (5mm,9mm),	RT 2 (4mm,5mm),	Symptoms reduced
35	Mrs.Rajeshwari	36/F	23-02-16	7- Weeks	Bilateral RT4 (3mm),LT(4mm)	USG Normal	Symptoms reduced
36	Mr.Deena dhayalan	38/M	24-02-16	8- Weeks	LT 5mm	USG Normal	Completed
37	Mr. Rajasekar	45/M	24-02-16	7- Weeks	RT 4.2mm	Stone expelled	Symptoms reduced
38	Mrs.Jeyanthi	42/F	29-02-16	7- Weeks	LT 4mm,	USG Normal	Completed
39	Mr.Viveganantha r	45/M	17-03-16	7- Weeks	RT 8 mm	3.1mm	Symptoms reduced
40	Mr Shanmugasundara m	46/M	21-03-16	7- Weeks	LT 5 mm	5mm	Symptoms relieved

LABORATORY INVESTIGATION REPORT

BEFORE TREATMENT

S.No	OP.No	Name	Age/ Sex	Haematological report							RFT			Urine Analysis		
				TC cells/ cu.mm	DC %			ESRmm		Hb gms %	Urea mg/dl	Creatinine mg/dl		Alb	Sug	Dep
					P	L	E	½ hr	1 hr							
1	5893	Mr. Karnan	37/M	9400	57	39	4	7	13	11	32	0.6		Nil	Nil	Opc
2	6705	Mr. Udhaya prakash	27/M	8400	54	42	4	6	15	10.2	31	1.0		Nil	Nil	Opc
3	8669	Mr. Ragavan	44/M	8000	57	39	4	5	15	12.6	34	0.9		Nil	Nil	Oec
4	271	Mr.Rjesh	25/M	9300	52	45	3	12	25	11	29	1.0		+	Nil	Opc
5	9055	Mrs.Anjammal	56/F	8900	60	37	3	7	12	10.2	29	0.8		Nil	Nil	Oec
6	3300	Mr.Vengateshan	31/M	9200	57	41	2	10	27	9.6	31	0.6		Nil	Nil	Nil
7	1546	Mrs.Lalitha	28/M	9200	60	36	4	10	26	11.9	29	0.9		Nil	Nil	Nil
8	1723	Mrs.Selvi	49/F	8800	61	35	4	6	15	10.2	24	1.0		Nil	Nil	Oec
9	2846	Mrs. Malliga	44/F	8400	53	41	6	15	26	13.4	26	0.5		Nil	Nil	Nil
10	2805	Mr.Dinesh kumar	20/M	8900	51	43	6	17	30	12.8	28	0.7		Nil	Nil	Opc
11	4176	Mr. Vasudevan	59/M	8300	60	35	5	10	22	8.7	27	0.8		Nil	Nil	Oec
12	9182	Mr.Mogamed	26/M	9600	59	38	3	17	25	13	29	1.2		Nil	Nil	Oec
13	2623	Mrs. Indira	31/F	8200	61	35	4	8	15	10.6	28	0.8		Nil	Nil	Oec
14	3288	Mrs.Amirthavalli	34/F	8300	59	37	4	6	12	9.9	26	0.7		Nil	Nil	Nil
15	3787	Mrs. Kanagavalli	52/F	8900	46	49	5	12	30	10.2	29	0.9		Nil	Nil	Oec
16	7792	Mr.Rajen	32/M	9000	56	39	5	9	16	13	27	0.9		Nil	Nil	Opc

17	5469	Mr.Sigamani	43/M	8800	55	42	3	11	25	13.5	22	0.9		+	Nil	Opc
18	9907	Mr. Amulraj	43/M	9000	51	44	5	19	25	11.9	27	0.6		Nil	++	Opc
19	9126	Mrs.Jeyabharathi	35/F	8900	60	37	3	10	22	13.8	26	0.9		Nil	Nil	Opc
20	1651	Mrs.Sundari	30/F	9200	58	39	3	8	16	10	30	1.1		Nil	Nil	Opc
21	2186	Mr.Jeyachandran	46/M	9400	56	39	5	11	27	14.5	29	0.8		Nil	+	Opc
22	5220	Mrs.Lalitha	30/F	9100	54	41	5	13	32	12.7	26	0.9		Nil	Nil	Opc
23	6456	Mrs. Bavya	35/F	8500	56	39	5	9	27	10.1	31	1.2		Nil	Nil	Oec
24	6455	Mrs.Anitha	33/F	7200	51	42	7	25	40	11.8	39	1.3		Nil	Nil	Oec
25	6880	Mr.Soundarrajen	31/M	6700	47	45	8	8	13	12.9	35	0.9		Nil	Nil	Oec
26	7582	Mrs. Rajeshwari	36/F	8000	52	42	6	25	49	12	36	1.8		Nil	Nil	Nil
27	9414	Mr. Rajaram	33/M	8200	53	42	5	17	29	14	31	1.4		Nil	Nil	Opc
28	9711	Mr.Kumar	37/M	7300	54	40	6	12	28	13	28	0.7		Nil	Nil	Opc
29	1727	Mr.Babu suresh	49/M	6500	51	44	5	7	15	10.1	28	1.2		Nil	Nil	Nil
30	1935	Mrs.Umamaheshwari	30/F	10000	60	36	4	19	37	12.6	35	1.1		Nil	Nil	Oec
31	4590	Mrs.Shanthi	52/F	8500	63	34	3	22	59	13.0	32	1.3		Nil	++	Opc
32	5736	Mrs.Geetha	52/F	9100	64	34	2	4	19	7.8	34	0.8		Nil	Nil	Opc
33	6226	Mr. Murali	44/M	8100	58	39	3	23	57	12.6	23	0.8		+	Nil	Nil
34	6912	Mr.Ranganathan	60/M	8500	63	34	3	18	43	10.7	24	1.0		Nil	Nil	Oec
35	7167	Mrs.Rajeshwari	36/F	9000	58	36	6	16	37	14.9	27	0.8		Nil	Nil	Nil
36	7498	Mr.Deena dhayalan	38/M	8700	60	36	4	2	9	8.9	37	1.0		Nil	Nil	Opc
37	7732	Mr. Rajasekar	45/M	9200	56	39	5	28	54	14.2	34	1.3		Nil	Nil	Opc
38	9213	Mrs.Jeyanthi	42/F	9000	58	40	2	13	43	12.9	30	0.8		Nil	Nil	Opc
39	4885	Mr.Vivegananthar	45/M	7900	56	42	2	6	21	14.9	35	0.9		Nil	+	Oec
40	6097	Mr.Shanmugasundaram	46/M	6300	63	32	5	16	29	15	26	0.7		Nil	Nil	Opc

TC – Total count, DC – Differential count, P – Polymorphs, L – Lymphocyte, E – Eosinophil, ESR – Erythrocyte Sedimentation Rate, Oe

LABORATORY INVESTIGATION REPORT

AFTER TREATMENT

S.No	OP.No	Name	Age/Sex	Haematological report							RFT		Urine Analysis		
				TC cells/ cu.mm	DC %			ESR		Hb gms %	Urea mg/ dl	Creatinine mg/dl	Alb	Sug	Dep
					P	L	E	½ hr	1 hr						
1	5893	Mr. Karnan	37/M	9500	59	36	5	6	13	11.8	27	0.7	Nil	Nil	Nil
2	6705	Mr. Udhaya prakash	27/M	8000	55	41	4	7	18	12	22	0.9	Nil	Nil	Nil
3	8669	Mr. Ragavan	44/M	8100	56	40	4	9	15	14.3	25	0.9	Nil	Nil	Nil
4	271	Mr.Rjesh	25/M	9500	59	37	4	10	23	12	25	0.8	Nil	Nil	Nil
5	9055	Mrs.Anjammal	56/F	8800	60	36	4	8	15	10.1	26	0.6	Nil	Nil	Oec
6	3300	Mr.Vengateshan	31/M	9300	57	40	3	3	8	10.4	29	0.9	Nil	Nil	Nil
7	1546	Mrs.Lalitha	28/M	7700	51	45	4	9	16	11..	27	0.8	Nil	Nil	Nil
8	1723	Mrs.Selvi	49/F	8100	64	32	4	7	15	11	28	1.0	Nil	Nil	Nil
9	2846	Mrs. Malliga	44/F	7000	64	31	5	5	14	10.4	27	0.8	Nil	Nil	Nil
10	2805	Mr.Dinesh kumar	20/M	8200	59	36	5	10	20	14.1	26	0.8	Nil	Nil	Nil
11	4176	Mr. Vasudevan	59/M	8500	63	34	3	11	25	8.9	28	0.9	Nil	Nil	Opc
12	9182	Mr.Mogamed	26/M	8200	56	40	4	7	13	15	28	0.6	Nil	Nil	Nil
13	2623	Mrs. Indira	31/F	9000	64	33	3	5	15	10.2	27	0.8	Nil	Nil	Nil
14	3288	Mrs.Amirthavalli	34/F	9300	56	39	5	12	19	11.8	28	0.9	Nil	Nil	Nil
15	3787	Mrs. Kanagavalli	52/F	8600	55	41	4	13	25	10.7	24	0.7	Nil	Nil	Opc
16	7792	Mr.Rajen	32/M	8000	62	35	3	4	9	12.2	21	0.8	Nil	Nil	Nil
17	5469	Mr.Sigamani	43/M	7900	56	40	4	7	15	12	24	0.8	Nil	Nil	Nil

18	9907	Mr. Amulraj	43/M	8400	63	34	3	9	12	11.1	23	0.7	Nil	+	Nil
19	9126	Mrs.Jeyabharathi	35/F	8800	57	40	3	8	17	11	28	0.9	Nil	Nil	Opc
20	1651	Mrs.Sundari	30/F	7200	57	39	4	5	13	14	29	0.9	Nil	Nil	Nil
21	2186	Mr.Jeyachandran	46/M	9300	51	45	4	5	12	13.4	21	0.6	Nil	+	Nil
22	5220	Mrs.Lalitha	30/F	8400	60	35	5	7	12	10.2	27	0.8	Nil	Nil	Nil
23	6456	Mrs. Bavya	35/F	8100	53	42	5	9	17	10.8	23	1.0	Nil	Nil	Nil
24	6455	Mrs.Anitha	33/F	7500	56	40	4	10	24	12.0	27	0.6	Nil	Nil	Opc
25	6880	Mr.Soundarrajen	31/M	7900	57	38	5	5	13	12.9	26	0.9	Nil	Nil	Nil
26	7582	Mrs. Rajeshwari	36/F	8400	59	38	3	15	25	10.4	32	1.3	Nil	Nil	Nil
27	9414	Mr. Rajaram	33/M	9600	58	38	4	13	21	14	30	1.0	Nil	Nil	Nil
28	9711	Mr.Kumar	37/M	7200	63	34	3	8	19	12	25	0.9	Nil	Nil	Nil
29	1727	Mr.Babu suresh	49/M	8200	56	38	6	6	10	10.8	27	0.8	Nil	Nil	Nil
30	1935	Mrs.Umamaheshwari	30/F	8900	60	35	5	5	14	12.9	22	1.0	Nil	Nil	Nil
31	4590	Mrs.Shanthi	52/F	8300	59	38	3	8	14	13.7	30	1.0	Nil	++	Opc
32	5736	Mrs.Geetha	52/F	8300	60	35	5	13	27	12.6	28	0.9	Nil	Nil	Nil
33	6226	Mr. Murali	44/M	9100	58	39	3	4	11	10.1	25	0.7	+	Nil	Nil
34	6912	Mr.Ranganathan	60/M	8100	60	37	3	11	26	10.4	27	0.9	Nil	Nil	Nil
35	7167	Mrs.Rajeshwari	36/F	6900	58	38	4	7	12	13.9	26	0.8	Nil	Nil	Nil
36	7498	Mr.Deenadayalan	38/M	8800	53	44	3	5	16	13.8	25	0.9	Nil	Nil	Nil
37	7732	Mr. Rajasekar	45/M	8200	50	45	5	7	17	10.6	23	0.8	Nil	Nil	Nil
38	9213	Mrs.Jeyanthi	42/F	9000	57	40	3	3	8	12.4	28	0.8	Nil	Nil	Oec
39	4885	Mr.Viveganantha r	45/M	8100	59	38	3	4	9	14.5	27	0.6	Nil	Nil	Nil
40	6097	Mr Shanmugasundaram	46/M	9300	53	44	3	15	28	15	29	0.8	Nil	Nil	Nil

– Occasional epithelial cells, Opc – Occasional pus cells, Alb – Albumin, Sug – Sugar, Dep – Deposits.

EXPELLED STONES

Arignar Anna Government Hospital
of Indian Medicine
Arumbakkam, Madras-600 106

APGHIM OP – 106

11/2/2016

Mr. Murali 44/m OP No 9951 Ayurvedic

USG whole abdomen

Liver – Mild fatty changes (+)

Gallbladder – Few calculus ng ~ 7 x 6 mm noted in gallbladder. Wall thickness (+) small polyps noted in gallbladder.

Pancreas & Retroperitoneum – obscured by bowel gas.

Spleen – (N)

P/D

Kidneys - E/o calculus ~ 4mm noted in lower pole of right kidney.

E/o clumped calculus ~ 13mm noted in midpole of left kidney.

urinary bladder - (N)

prostate - mildly enlarged in size (vol ~ 31ml)

Liver - - Gallbladder calculus

- Bilateral renal calculi -
- Mild prostatomegaly
- Mild fatty liver

SWAMI VIVEKANANDA DIAGNOSTIC CENTRE

Lions Edifice for Service Trust Complex, Inside D.G.Vaishnav College
Chennai - 600 106. PH: 044 - 2363 7521 / 2363 7604

atient name	13MURALI GOPALA ACHAR	Age/Sex	44/m
atient ID	13_05_2016_14_03_36	Visit No	1
eferred by	Cyll	Visit Date	13/05/2016

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Chennai-600 106. PH : 044 - 2363 7521 / 2363 7604

Patient name	MR. MURALI GOPALA ACHAR	Age/Sex	44 Years / Male
Patient ID	2658	Visit No	1
Referred by	Dr. GH	Visit Date	13/05/2016

Abdomen and KUB Scan Report

Real time B-mode Ultrasonography of Abdomen and KUB done

Abdomen

Liver filled with homogeneous parenchymal echoes. No abscess or mass lesion in the liver

Gall bladder appeared normal. No calculi seen in the gall bladder

Commonduct appeared normal. No calculi seen in the commonduct.

Pancreas appeared normal

Spleen appeared normal

Aorta appeared normal. No para aortic nodes seen.

Peritoneal cavity appeared normal

KUB

Right kidney measured 9.7 X 3.9 cms.

Cortex and collecting system of right kidney appeared normal. No calculi seen.

Left kidney measured 9.1 X 5.0 cms.

Cortex and collecting system of left kidney appeared normal. No calculi seen.

Bladder appeared normal

Prostate measured 4.8 X 3.1 X 2.8 cms. (Weight = 21.67 gms.)

Prostate appeared normal. No intra vesical enlargement of prostate gland seen.

Impression

Normal study

DR.R.KANAGASABAI.,MD.,DMRD
CONSULTANT RADIOLOGIST

Dr. R. KANAGASABAI, MD, DMRD
CONSULTANT RADIOLOGIST
REGN. No. 45793

DISCUSSION

DISCUSSION

KALLADAIPPU is a common disease pertaining to the kidney. Large populations are suffering from this disease. But they are not completely relieved from their symptoms by other systems of medicine. Hence with the help of trial medicine from Siddha system, results and observations are noted for this study.

The patients were examined base on Siddha and as well as modern aspects. All the necessary investigations were made during the study. The results obtained from their studies were discussed below for better conclusion.

Trial medicine administered was vediyuppu chendooram – 130-260mg 2 times a day with mullangi kizhangu chaaru after food for 48 days.

1. **Sex distribution:**

   Among 40 cases 23 were males and 17 were females.

2. **Age distribution**:

   Although all the decades of people are affected 35% were affected in 3^{rd} decade, 30% were affected in 4^{th} decade.

3. **Occupation** :

   40% were affected in office worker, 22.5% were affected in Farmer, 20% were affected in House wife.

   Mixed categories of people are affected, from housewife to working woman,

   students to retired person. House wives and office workers were affected commonly.

4. **Food habits**:

   Among 40 patients, 10 patients eat vegetarian food and others eat mixed diet.

5. **According to season**:

   The highest incidence were noted in pinpani kaalam (52.5%) and 17.5% were noted in munpani Kaalam. This shows that due to hot climate, majority of the people have been reported during this period.

6. **Distribution of thinai**:

According to thinai, the highest distribution 70% was noted in neithal, 15% in Marutham and 12.5% noted in kurinji.

7. **On clinical manifestations:**

All of my patients were present one or more urinary symptoms, 100% were having flank pain, 50% were having burning Micturition,20% were having dysuria, 7.5% had nausea and hematuria, 5% had Oliguria and 2.5% had vomiting and urine retention.

8. **Mukkutram:**

Distribution of vatham:

Among the patients 100% were affected in Abanan,Viyanan and Samanan, 30% in Devathathan, 20% in Kirukaran and 10% in Koorman and udhanan.

Affected Abanan produced burning Micturition, constipation and hematuria.

Affected viyanan produce difficulty in movements.

Affected Samanan produced indigestion.

Affected Uthanan produced nausea and vomiting.

Affected Koorman produced impairment of eye sight

Affected Kirukaran produced loss of appetite.

Affected Devathathan produced loss of

Distribution of pitham:

Among the treated patients 100% were affected in Saathagam, 22.5% were affected in Ranjagam, 12.5% in Analagam, and 10% were affected in alosagam.

The affected Analagam produced loss of appetite.

Affected Ranjagam produced pallor of skin, eye and reduced hemoglobin.

Affected Saathagam produced difficulty in doing routine work

Affected Alosagam produced impairment of eye sight

Distribution of Kabham:

Among the patients, 45% were affected in Santhigam, 12.5% affected in kiletham, 10% affected in pothagam and tharpagam, 2.5% affected in Avalambagam.

Affected Klethagam produced loss of appetite.

Affected Santhigam produced low back pain, knee joint pain.

Affected pothagam produced loss taste.

Affected tharpagam produced impairment of eye sight.

Affected avalambagam produced dyspnea.

9. Ezhu Udal Thathukkal:

Among the treated patients saram was affected in 100% of patients, Enbu was affected in 32.5% and senheer in 25%, oon was affected in 2.5%.

10. Enn vagai thervugal:

In this, Moothiram was affected in 65%, Mozhi was affected in 25%, Sparisam in 12.5%, Malam in12.5% and Vizhi in 10%, Naa was affected in 5%

11. Naadi:

On examination of naadi, 30% had vazhiazhal naadi, 5% had vali Iyam, 52.5%% had azhal vali naadi, 5% had kabha vatha naadi and 7.5% had kabha pitha naadi.

12. Neikuri:

On Neikuri examination 55% were having vatha neer, 30% were having pitha neer and 15% were having kabha neer.

A drop of gingely oil dropped into the early morning urine sample in a bowl may result in spread like snake called vatham, like a ring in pitham, like pearl in kabham.

13. Urinary calculi based on location:

In total 40 patients 20 were having bilateral stones (50%), 13 were having right side stone (32.5%) and 5 were having left side stone (12.5%), 2 were having ureteric calculi.

This shows that the ureteric calculi are easily pushed out by Allopathic treatments and the people with calculi in the kidney approaches siddha system for a permanent relief.

14. Special investigation:

USG- abdomen and pelvis is advised for all the patients to confirm the diagnosis.

After confirming the diagnosis, the patients were given the trial medicine and instructed to follow the diet and other restrictions based on Siddha system.

15. Urine analysis:

It was observed that, 40% of cases showed pus cells in urine.

16. Mode of action of the drug:

According to suvai:

The trial medicine Vediyuppu chendooram has astringent taste.

This taste will equalize the increased pitham which is the main cause for Kalladaippu. This medicine acts against the increased pitham. So it is considered as Ethirurai maruthuvam.

According to veeriyam: (nature)

The trial medicine Vediyuppu chendooram possesses thatpa veeriyam. So it cures pitha diseases. By this Vediyuppu chendooram treats Kalladaippu noi.

17. Bio chemical analysis:

Vediyuppu chendooram has Chloride, magnesium, Flouride and iron.

18. Toxicological analysis:

Acute and Sub acute toxicity studies were conducted at Baid metha College of Pharmacy. At the end of toxicity studies the hematological parameters (TC, DC and Hb),

Biochemical parameters (LFT, KFT) and histopathology of vital organs like Liver, Kidney, Spleen and Lungs were carried out. Vediyuppu chendooram shows no toxic effect.

19. Pharmacological analysis:

Pharmacological studies of the trial medicine Vediyuppu chendooram showed Lithotriptic actions in albino wistar rats.

The results of preclinical screening, the results of Chemical analysis, Toxicological studies, Pharmacological studies are shown in annexures.

20. Statistical analysis:

The preclinical studies of trial medicine Vediyuppu chendooram statistically analysed and showed significant result.

Statistical analysis of clinical study were done for the subjective and objective parameter, observed before and after treatment statistical results of preclinical and clinical study were attached to annexure.

21. Results after treatment:

Many of the patients were relieved of their problem, 60% showed good result that is both by symptoms and by Sonographic findings, 25% showed moderate result who are relieved of their symptoms and reduction in stone size, 15% showed poor result were not relieved of their problem.

SUMMARY

SUMMARY

- I like to summarize this study with the following highlights.
- Males are more prone to get kalladaippu than females according to my studies.
- In age distribution, 3rd and 4th decades of people are more affected.
- Office workers and House wives occupy the first two places in occupational classification.
- Most of the patient had mixed diet.
- Higher incidence of cases were noted in pinpani kalam.
- In the disturbance of Ezhu udal thathukkal, 100% were affected by saaram, 32.5% were affected by enbu and 25% were affected in seneer.
- In Naadi, most of the patients having 52.5% in azhal vali naadi, 30% in vali azhal naadi.
- In Neikuri examination 55% were having vatha neer.
- Among the patients, 100% had flank pain, 50% had burning Micturition, and 20% had Dysuria.
- 50% patient had bilateral renal calculi, 32.5% patient had right renal calculi.
- All of my patients were administered with my trial medicine Vediyuppu chendooram– 130-260 mg bd with mullangi kizhangu chaaru after food for a period of 48 days.
- After treatment with this trial medicine, most of the symptoms like loin pain, burning Micturition and Dysuria are relieved. The trial medicine shows 60% good result and 25% moderate result and 15% poor result.

CONCLUSION

CONCLUSION

- Kalladaippu is a common disorder of pitha kutram. The dearranged pitham is settled down by the trial medicine having astringent taste thereby the medicine acts as ethirurai maruthuvam to cure the disease.
- Most of the cases noted in pinpani kalam and munpani kalam in my clinical trial. So, people should take all preventive measures during this period and take enough water.
- Toxicological study shows no acute and sub acute toxicity.
- Pharmacological study reveals that the trial medicines possess lithotriptic activity.
- During clinical trial, no adverse reactions or complications were observed.
- The palatability of the trial drug is astringent, so it is easier to consume to the patients.

The trial medicine Vediyuppu chendooram showed good results with relieving symptoms in almost 85% patients.

ANNEXURE – I

CERTIFICATES

INSTITUTIONAL ETHICAL COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref: Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
DR.P.JEYAPRAKASH NARAYANAN M.D(S)., Chairman		
DR.V.BANUMATHI M.D(S)., Member Secretary		
DR.N.KABILAN M.D(S)., Clinician- Siddha	✓	
DR.P.SATHIYA RAJESWARAN M.D(S)., Clinician- Siddha	✓	
DR.G.AADINATH REDDY,M.Pharm, Ph.D., Pharmacologist	✓	
DR.S.THILAGAVATHY Msc.,Ph.D., Social Scientist	✓	
DR.T.MAHALAKSHMI M.A.,Ph.D., Linguistic Expert	✓	
DR.P.VIDYA M.B.B.S., DMRD., Modern Medicine Expert	✓	
MR.P.SARAVANAN., Public Person	✓	

13/6/14.

Dr.P.Jeyaprakashnarayanan M.D(S)., Chairman

Dr.V.Banumathi M.D(S)., Member Secretary

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethical Committee (IEC)

IEC No: GSMC-CH-ME-3/001/2014

Protocol title: AN OPEN CLINICAL STUDY ON KALLADAIPPU (UROLITHIASIS) WITH THE EVALUATION OF SIDDHA DRUG VEDIYUPPU CHENDOORAM	
Principal Investigator:	DR.P.GOVINDAMMAL
Name & Address of Institution: Government Siddha Medical College, Arumbakkam, Chennai-106	
☐ New Review	☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY):	13-06-2014
Date of Previous Review, If Revised Application:	
Decision of the IEC	
☑ Recommended	☐ Recommended with suggestions
☐ Revision	☐ Rejected
Suggestions / Reasons / Remarks: (i) In primary outcome, add reduction in the no of stones, size of stones, expulsion of stones (ii) Instead of subacute toxicity can do repeated 28 days oral toxicity studies.	
Recommended for a period of 1 year from date of completion of preclinical studies :	

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IEC approval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

13/6/14

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V.Banumathi
Member Secretary

Jaya Indian Medicine Pharmaceutical (P) Ltd

89, Velnagar II Street, Maduravoyal, Chennai - 600 095. Ph : +91 44 6516 5825
Mob (O): +91 92822 36113 E-mail : jayasai.siddha@gmail.com

SIDDHALAYA
Siddha way to a Healthy Life

Recognize

Dr. P. GOVINDAMMAL, MD(s)

for participating in the symposium on "Siddha System of Medicine raising to modern trends" on Sunday, the 10th day of April 2016 at World University Service centre 18E, Spur tank road,chetpet, Chennai-600031.

Prof. Dr. P. Jeyaprakash Narayanan., MD
Chairman and Managing Director

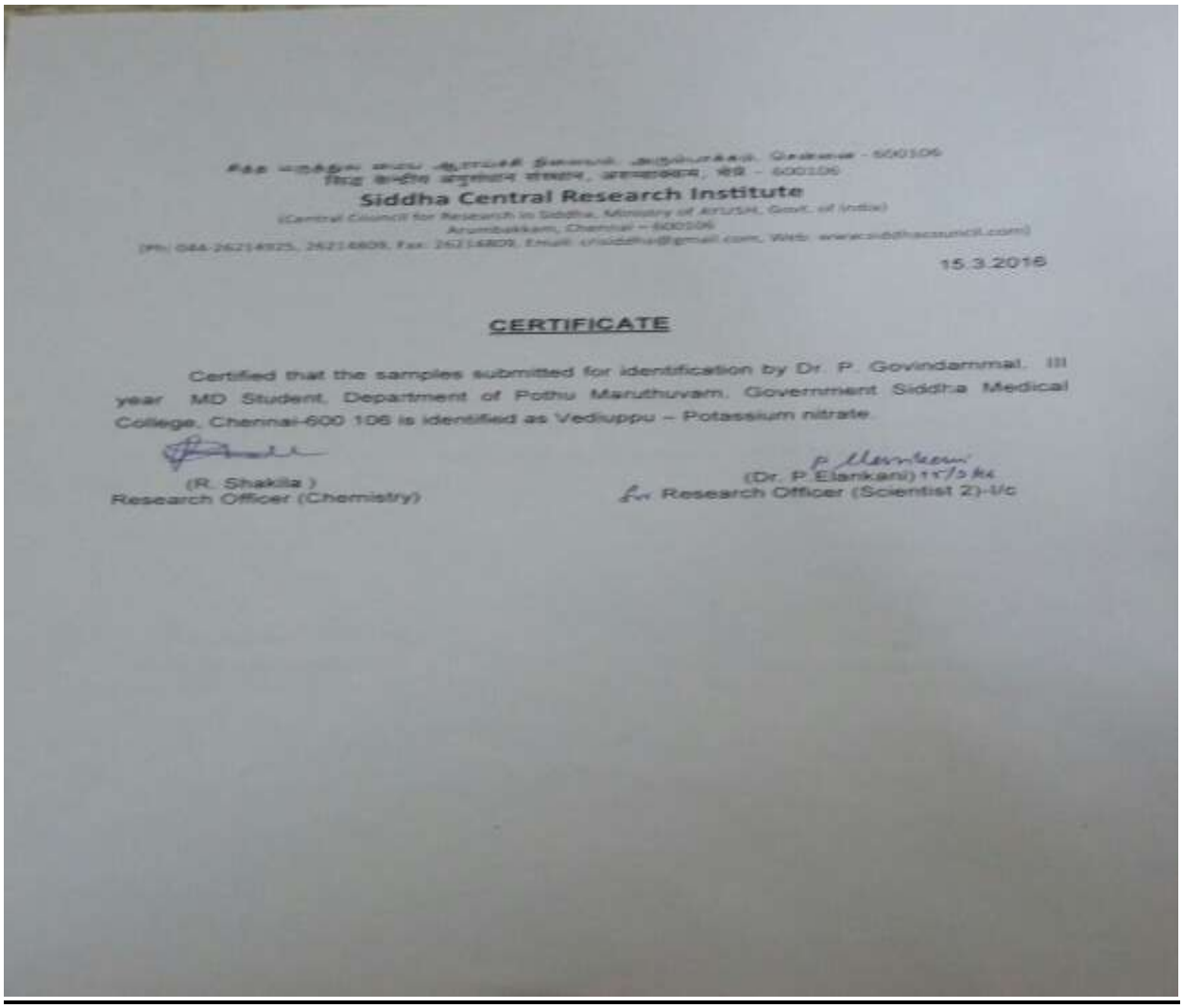
Certificate

This is certify that the project titledToxicological and pharmacological activity of VEDIYUPPU CHENDHOORAM in ratshas been approved by the

IAEC No: IAEC/XLIV/18/CLBMCP/2014

Name of Chairman/ Member Secretary IAEC:

Signature with date



Siddha Central Research Institute

(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)

Arumbakkam, Chennai – 600106

15.3.2016

CERTIFICATE

Certified that the samples submitted for identification by Dr. P. Govindammal. III year MD Student, Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106 is identified as Vediuppu – Potassium nitrate.

(R. Shakila)
Research Officer (Chemistry)

(Dr. P. Elankani)
for Research Officer (Scientist 2)-I/c

சித்த மருத்துவ ஆய்வுப்பிரிவு நிலையம், அரும்பாக்கம், சென்னை - 600 106
सिध्द केन्द्रीय अनुसंधान संस्थान, अरुम्बाक्कम, चेन्नई- 600 106
Siddha Central Research Institute
Arignar Anna Govt. Hospital Campus, Arumbakkam, Chennai-600 106
(Central Council for Research in Siddha, Department of AYUSH,
Ministry of Health & Family Welfare, Govt. of India)
Phone: 044-26214925, Tele Fax: 044-26214809, E-mail: crisiddha@gmail.com, Web: www.crisiddha.tn.nic.in

22nd February 2016

CERTIFICATE

Certified that the leaf submitted for identification by Dr. P. Govindammal, PG III year, Department of Pothu maruthuvam, Government Siddha Medical College, Arumbakkam, Chennai - 600 106, is identified as Vetrilai – *Piper betle* L. (Leaf).

Sasikala Ethirajulu
Consultant (Pharmacognosy)

22/2/16
P.Sathiyarajeswaran
Assistant Director Incharge

The Tamil Nadu Dr.M.G.R. Medical University

#69, Anna salai, Guindy, Chennai-600 032.

This certificate is awarded to

Dr./~~Mr./Ms.~~ GOVINDAMMAL . P

for participating as ~~Resource Person~~ / Delegate in the Fourteenth Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post Graduates & Researchers

Organised by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University from 5th to 9th May 2014.

Dr. N. KABILAN M.D. (Siddha)
Reader, Dept. of Siddha

Dr. JHANSI CHARLES, M.D.
Registrar

Prof. Dr. D. SHANTHARAM, M.D., D.Diab.,
Vice-Chancellor

C.L.BAID METHA COLLEGE OF PHARMACY

(An ISO 9001-2000 certified institute)

Jyothi Nagar, Old Mahabalipuram Road

Thoraipakkam, Chennai – 600 097

CERTIFICATE

This is to certify that the project entitled, **Toxicological and Pharmacological study on VEDIYUPPU CHENDHOORAM** in rats submitted in partial fulfilment for the degree of **M.D. (siddha)** was carried out at C.L. Baid Metha college of Pharmacy, Chennai-97, in the Department of Pharmacology during the academic year of 2014-2015.

(Dr.P.Muralidharan)

Mr. P.Muralidharan, M.Pharm, Ph.D
Professor and Head
Department of Pharmacology,
C.L.Baid Metha college of pharmacy,
Chennai-97

ANNEXURE – II

BIO – CHEMICAL ANALYSIS

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
B	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	white precipitate obtained	Present
3	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Absence of Yellow precipitate	Abscent
4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white Precipitate	Absent
5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hcl.	Absence of Rotten egg smelling	Absent
6	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube	Absence of reddish brown gas.	Absent

	vertically down.		
7a	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white Precipitate	Absent
B	5 drops of clear solution is added with 2ml of diluted sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	KMNO4 solution Discolourisation obtained	Present
8	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops of Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent
9	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution.	Absence of Yellow Precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absence of Bluish green coloured flame.	Absent
B	2ml of the extract is added with excess of Ammonia solution	Absence of deep Blue	Absent
12	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess	Absence of White Precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate Solution is added.	Absence of Blood red colour	Absent
B	To the 2 ml of extract, 2 ml of	Blood red colour	Present

	Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	obtained	
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
15	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Absence of White precipitate.	Absent
16	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown Precipitate	Absent
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absence of Yellow precipitate	Absent
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow Precipitate	Absent
21	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution.	Absence of Yellow precipitate	Absent
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil	Absence of Green colour	Absent

	for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.		
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium lodide solution.	Absence of Red colour	Absent
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH, mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

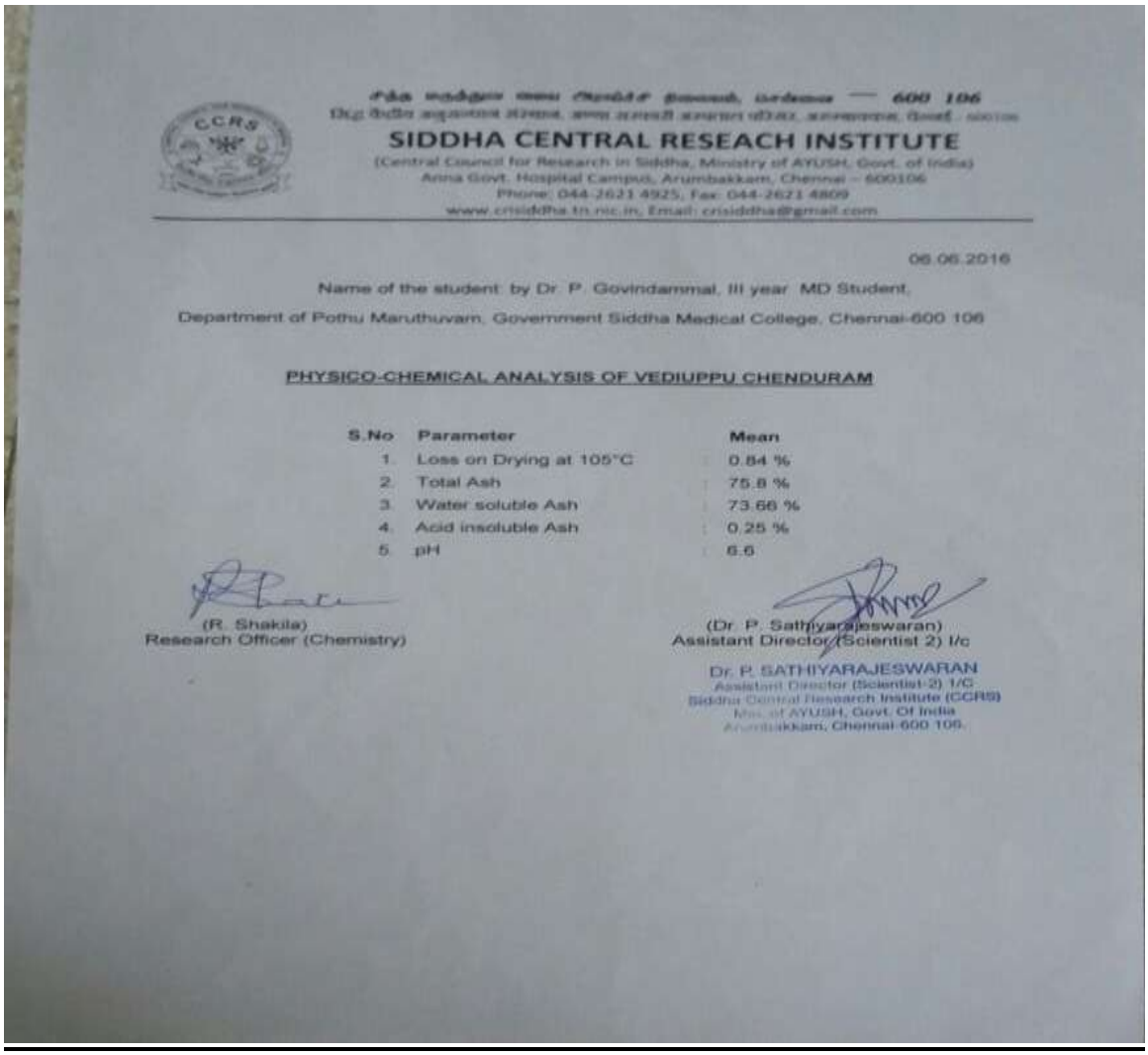
The given sample (vediyuppu chendooram) contains

Chloride

Flouride

Iron.

PHYSICO CHEMICAL ANALYSIS



சித்த மருத்துவ ஆய்வு மையம், சென்னை — 600 106
மத்திய இந்திய மருத்துவ ஆராய்ச்சி மன்றம், ஆயுஷ் அமைச்சகம், இந்திய அரசு

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06.06.2016

Name of the student by Dr. P. Govindammal, III year MD Student,
Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106

PHYSICO-CHEMICAL ANALYSIS OF VEDIUPPU CHENDURAM

S.No	Parameter	Mean
1.	Loss on Drying at 105°C	0.84 %
2.	Total Ash	75.8 %
3.	Water soluble Ash	73.66 %
4.	Acid insoluble Ash	0.25 %
5.	pH	6.6

(R. Shakila)
Research Officer (Chemistry)

(Dr. P. Sathiyarajeswaran)
Assistant Director (Scientist 2) I/c

Dr. P. SATHIYARAJESWARAN
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Siddha Central Research Institute (CCRS)
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Arumbakkam, Chennai-600 106.

ANNEXURE – IV

TOXICOLOGICAL STUDY

ACUTE ORAL TOXICITY – OECD GUIDELINES - 423

Acute toxicity study was carried out as per OECD guideline (Organization for Economic Co - operation and Development, Guideline-423

Animal : Healthy wistar albino female rat weighing 200–220 gm

Studied carried out at three female rat under fasting condition, signs of toxicity was observed for every one hour for first 24 hours and every day for about 14 days from the beginning of the study.

INTRODUCTION:

The acute toxic class method is a stepwise procedure with the use of 3 animals of a single sex per step. Depending on the mortality and/or the moribund status of the animals, on average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. Morbid animals or animals obviously in pain or showing signs of severe and enduring distress shall be humanely killed, and are considered in the interpretation of thetest results in the same way as animals that died on test. The method allows for the determination of an LD50 value only when at least two doses result in mortality higher than 0% and lower than 100%.

PRINCIPLE:

It is the principle of the test that based on a stepwise procedure with the use of a minimum number of animals per step, sufficient information is obtained on

the acute toxicity of the test substance to enable its classification. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex. Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.; − no further testing is needed − dosing of three additional animals with the same dose − dosing of three additional animals at the next higher or the next lower dose level. The method will enable a judgment with respect to classifying the test substance to one of a series of toxicity classes.

METHODOLOGY

Selection of animal species:

The preferred rodent species is rat, although other rodent species may be used. Healthy young adult animals of commonly used laboratory strain Swiss albino is used . Females should be nulliparous and non-pregnant. Each animal at the commencement of its dosing should be between 8 and 12 weeks old and its weight should fall in an interval within±20 % of the mean weight of the animals.

Housing and feeding conditions:

The temperature in the experimental animal room should be 22ºC (+3ºC). Although the relative humidity should be at least 30% and preferably not

exceed 70% other than during room cleaning the aim should be 50-60%. Lighting should be artificial, the sequence being 12 hrs light, 12 hrs dark. For feeding, conventional laboratory diets may be used with an unlimited supply of drinking water. Animals may be grouped and tagged by dose, but the number of animals per cage must not interfere with clear observations of each animal.

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 7 days prior to dosing to allow for acclimatization to the laboratory conditions.

Observation done:

Group	Day
Body weight	Normal
Assessments of posture	Normal
Signs of Convulsion Limb paralysis	Absence of sign (-)
Body tone	Normal
Lacrimation	Absence
Salivation	Absence
Change in skin color	No significant colour change
Piloerection	Normal
Defecation	Normal

Sensitivity response	Normal
Locomotion	Normal
Muscle gripness	Normal
Rearing	Mild
Urination	Normal

Dose mg/kg	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
2000	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-

1.*Alertness 2.Aggressiveness 3. Pile erection 4. Grooming 5.Gripping 6. Touch Response 7. Decreased Motor Activity 8.Tremors 9 Convulsions 10. Muscle Spasm 11. Catatonia 12.Musclerelaxant 13.Hypnosis 14.Analgesia15.Lacrimation 16. Exophthalmos 17. Diarrhea 18. Writhing 19 Respiration 20. Mortality*

Acute toxicity:

In the acute toxicity study, the rats were treated with different concentration of shoot extract of Vediyuppu chendooram from the range of 5mg/kg to 2000mg/kg which did not produce signs of toxicity, behavioral changes, and mortality in the test groups as compared to the controls when observed during 14 days of the acute toxicity experimental period. These results showed that a single oral dose of the extract showed no mortality of these rats even under higher dosage levels indicating the high margin of safety of this extract. In acute toxicity test the extract of Vediyuppu chendooram was found to be non toxic at the dose level of 2000mg/ kg bodyweight.

Sub-Acute toxicity test

The dose selected for the sub acute toxicity study was 100mg, 200mg/kg of Vediyuppu chendooram. All the animals were free of intoxicating signs throughout the dosing period of 28 days. No physical changes were observed throughout the dosing period. No mortality was observed during the whole experiment. No abnormal deviations were observed. No significant changes were observed in the values of different parameters studied when compared with controls and values obtained were within normal biological and laboratory limits. The weights of organs recorded did not show any significant differences in the treatment and the control group indicating that Vediyuppu chendooram was not toxic to kidney, liver and spleen There was no significant changes were observed in hemoglobin (Hb), red blood cell (RBC), white blood cell (WBC), packed cell volume (PCV), Erythrocyte sedimentation rate (ESR) in all the treated groups as compared to respective control groups. Histopathology studies were carried out on liver, kidney and spleen and recorded

SUB ACUTE TOXICITY REPORTS

Vediyuppu chendooram 100 mg/kg

HAEMOTOLOGY

CBC

WBC : 10,000 cells/cumm

Differential Count

NEUTROPHILLS : 10%

LYMPHOCYTES : 89 %

EOSINOPHILS : 01 %

MONOCYTES	:	00 %
RBC	:	8.78 millions/cumm
HB	:	16.0 gms%
PCV	:	50.3 %
MCV	:	57.3 fL
MCH	:	18.2 pg
MCHC	:	31.8 Grams/dl
PLATELET	:	4.8 Lakhs/cumm

BIOCHEMISTRY

Blood sugar	: 89 mg/dl
BUN	: 50.9 mg/dl
Creatinine	: 0.9 mg/dl
SGOT	: 82 U/L
SGPT	: 59 U/L
ALP	: 140 U/L
T.Protein	: 8.0 grams/dl
Albumin	: 3.9 grams/dl

LIPID PROFILE

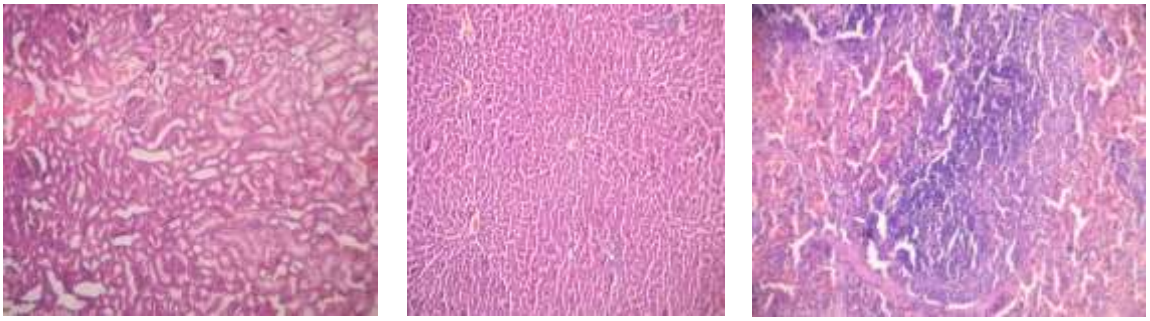
T. Cholesterol	: 105 mg/dl
Triglycerides	: 68 mg/dl
HDL	: 22 mg/dl
LDL	: 69.4 mg/dl
VLDL	: 13.6 mg/dl

Ratio 1(T.CHO/HDL) : 4.77

Ratio 2(LDL/HDL) : 3.1

Histopathological report

Kidney Liver Spleen



Vediyuppu chendooram 200 mg/kg

HAEMOTOLOGY

CBC

WBC	:	5,200 cells/cumm
Differential Count		
NEUTROPHILLS	:	10%
LYMPHOCYTES	:	89 %
EOSINOPHILS	:	01 %
MONOCYTES	:	00 %
RBC	:	8.98 millions/cumm
HB	:	16.9 gms%
PCV	:	53.6 %
MCV	:	59.7 Fl
MCH	:	18.8 pg
MCHC	:	31.5 Grams/dl
PLATELET	:	7.20 Lakhs/cumm

BIOCHEMISTRY

Blood sugar	:	90 mg/dl
BUN	:	52.8 mg/dl
Creatinine	:	0.9 mg/dl
SGOT	:	72 U/L
SGPT	:	49 U/L
ALP	:	135 U/L
T.Protein	:	8.1 grams/dl

Albumin : 3.1 grams/dl

LIPID PROFILE

T. Cholesterol : 112 mg/dTriglycerides : 69 mg/dl

HDL : 26 mg/dl

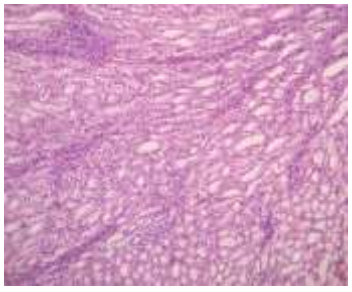
LDL : 72.2 mg/dl

VLDL : 13.8 mg/dl

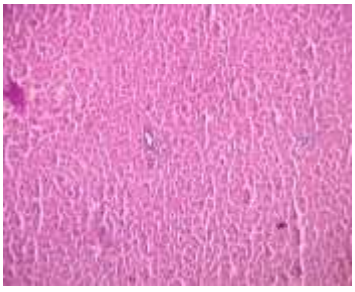
Ratio 1(T.CHO/HDL) : 4.30

Ratio 2(LDL/HDL) : 2.77

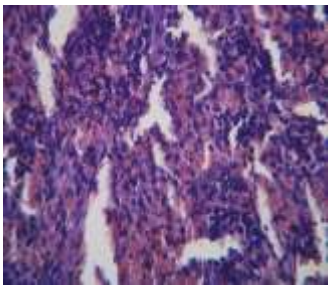
Kidney



Liver



Spleen



ANNEXURE – V

PHARMACOLOGICAL STUDY

ANTI UROLITHIASIS ACTIVITY

Stone induction–Kidney stones were induced by 0.75% of ethylene glycol in drinking water for 28 days ad libitum. After 28 days induction, the animals were used for the study

Experimental design:

Groups	**Treatment**
Group I	Normal Control
Group II	Urolithiasis rats- 0.75% of ethylene glycol for 28 days
Group III	Urolithiasis rats + Vediyuppu chendooram 200mg/kg for 28 days
Group V	Urolithiasis rats + Vediyuppu chendooram 400mg/kg for 28 days

In the experiment a total of 24 rats (6 normal rats, 18 urolithic rats) were used. The rats were divided into 4 groups of 6 rats each. Group I (Gr I)-Normal control rats; Group II (Gr II)-Urolithic rats, (Gr III)-Urolithic rats given Vediyuppu chendooram (200mg/kg, p.o.for 28 days) and (Gr IV)-Urolithic rats given Vediyuppu chendooram (400mg/kg, p.o.for 28 days). At the end of 28 days, the animals were housed in metabolic cages and collected urine after 24 h.

Biochemical assays

The urine collected after 24 h was subjected to analyze calcium [1], oxalate [2], uric acid [3,], citrate [4],

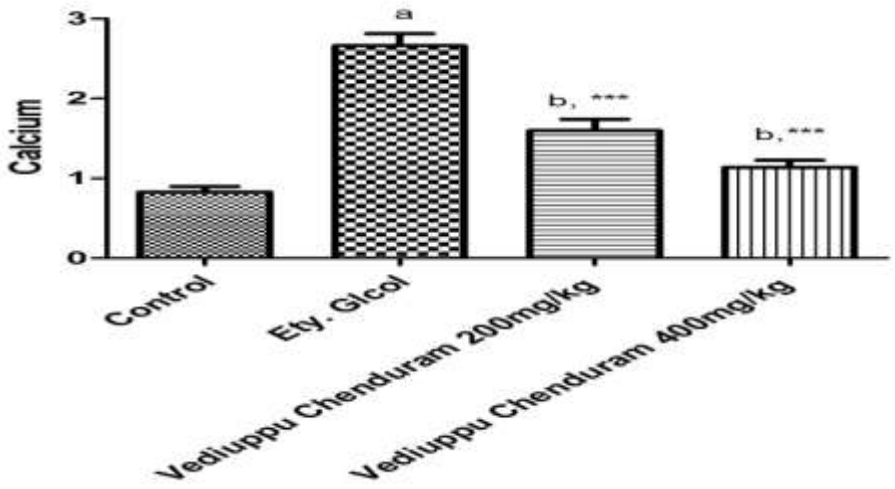
REFERENCES

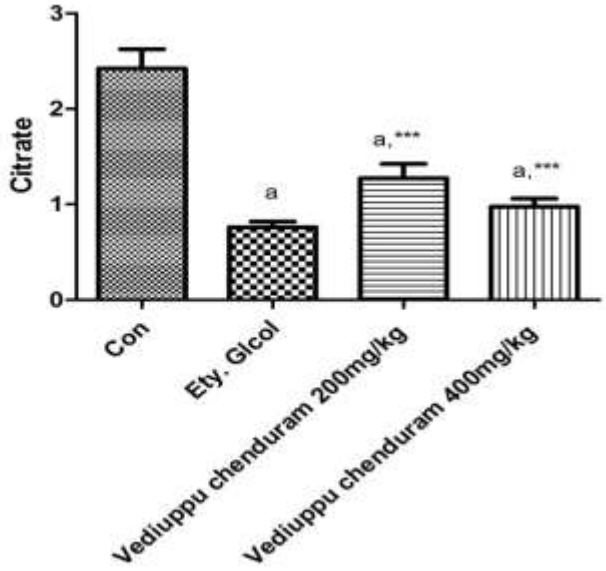
1. Gitelman H J, An improved automated procedure for determination of calcium in biochemical specimen. Anal Biochem, 18 (1967) 521.
2. Hodgkinson A & Williams H E, An improved colorimetric procedure for urine oxalate. Clin Chem Acta, 36 (1972) 127.
3. Caraway W T & Seligson, in Standard methods of clinical chemistry, (Academic Press, New York) 1963, 239.
4. Bergmeyer H U, Citrate, in Methods in enzymatic analysis, volume 7, 3rd edition, edited by Bergmeyer, Grabl, (VCH Verlagsgesellschatt, Germany) 1985, 2.

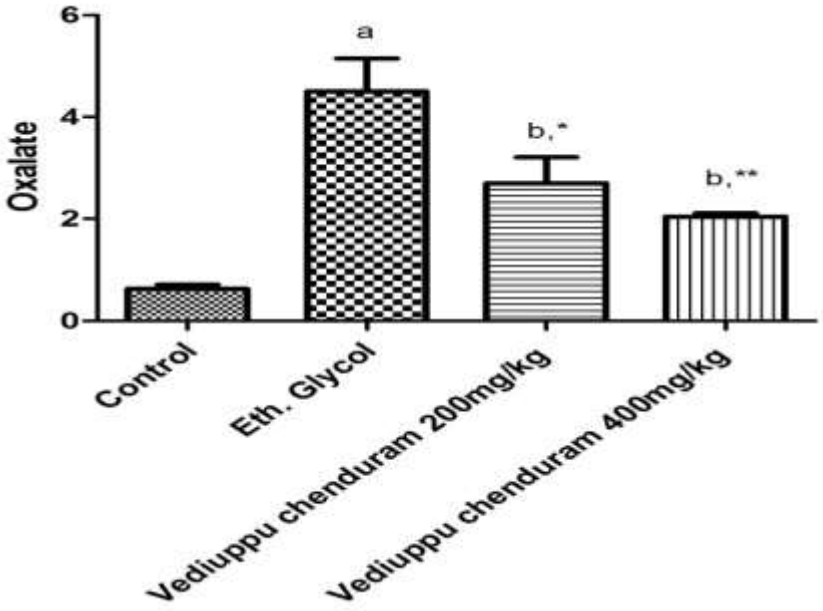
Effect of Vediyuppu chendooram on ethylene glycol induced urothiasis activity in rats

Biochemical Assays	Gr-I	Gr-II	Gr-III	Gr-IV
Calcium	0.82±0.07	2.66±0.15[a]	1.60±0.14[b] ***	1.14±0.08 b ***
Oxalate	0.62±0.08	4.50±0.64 [a]	2.70±0.50 [b] *	2.05±0.06 [b] **
Uric acid	0.82±0.04	2.75±0.30 [a]	2.20±0.37 [a]*	1.42±0.25 [b] *
Citrate	2.42±0.20	0.76±0.05 [a]	1.27±0.14 [a]***	0.97±0.08[a]***

Calcium, oxalate, citrate, uric acid, mg/24 h urine. Superscript letters represent P <0.05 (Tukey's test). [a] As compared with group I, [b] As compared with group II.*P<0.05, **P <0.01, ***P<0.001.


Calcium
3
2
1
0
a
b, ***
b, ***
Control
Ety. Glcol
Vediuppu Chenduram 200mg/kg
Vediuppu Chenduram 400mg/kg


Citrate
3
2
1
0
a
a,***
a,***
Con
Ety. Glcol
Vediuppu chenduram 200mg/kg
Vediuppu chenduram 400mg/kg


Oxalate
6
4
2
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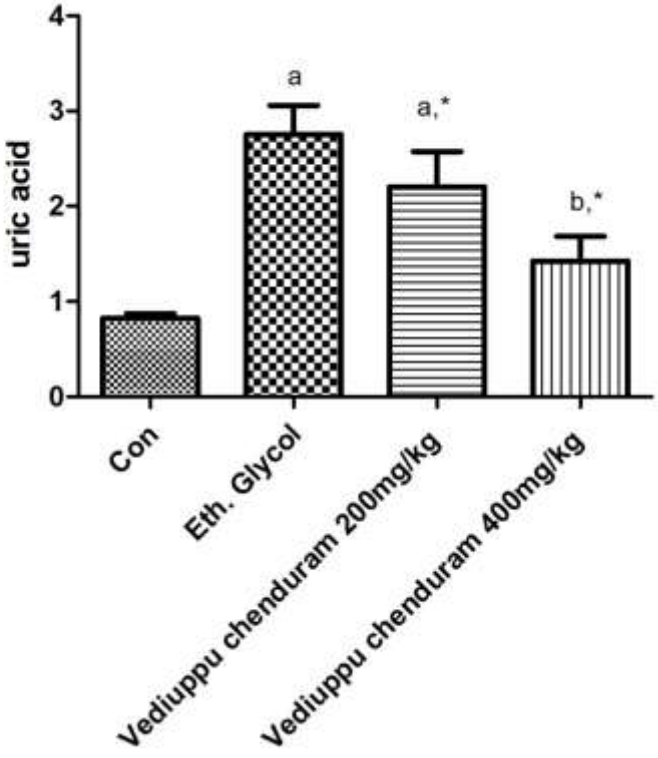
a
b,*
b,**
Control
Eth. Glycol
Vediuppu chenduram 200mg/kg
Vediuppu chenduram 400mg/kg


uric acid
4
3
2
1
0
a
a,*
b,*
Con
Eth. Glycol
Vediuppu chenduram 200mg/kg
Vediuppu chenduram 400mg/kg

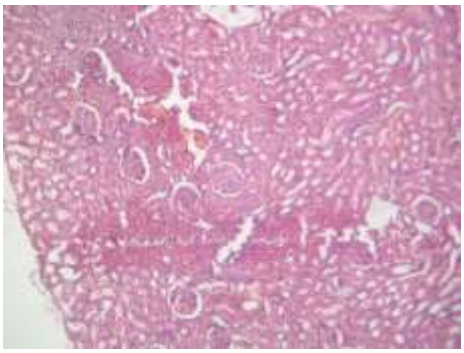
Histopathology:

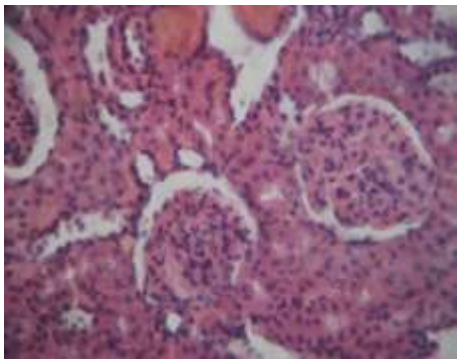
Group I- Kidney of control rats showing normal cellular structure. H and E × 100

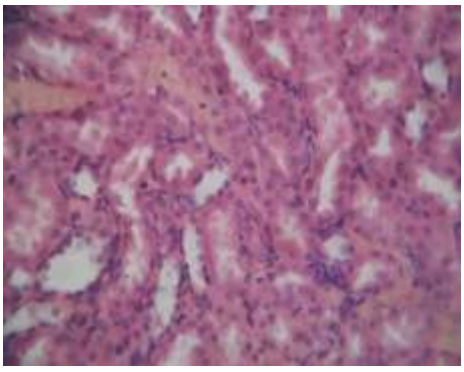
Group II- Kidney of ethylene glycol induced urolithic rats showing magnified view of calcium oxalate stone with highly refractive crystal deposits

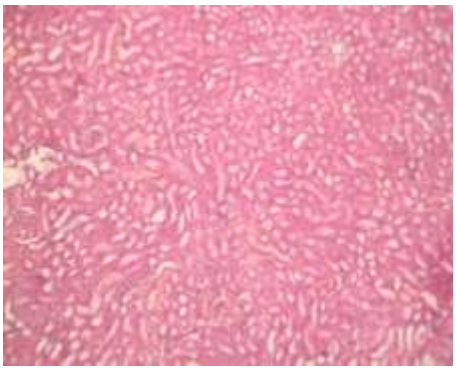
Group III- Vedippu Chenduram 200 mg/kg treated rats showing near normal renal structure. H and E × 100.

Group IV- Vedippu Chenduram 400 mg/kg treated rats showing normal renal architecture. H and E × 100









Group 1:

ANNEXURE – VI

BIO STATISTICAL ANALYSIS

BIO STATISTICAL ANALYSIS

CLINICAL PROGNOSIS

Treatment for Kalladaippu:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs&Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Flank pain	40(100)	9(22.5)**
2.	Nausea	3(7.5)	1(2.5)*
3.	Vomiting	1(2.5)	0(0)*
4.	Burning Micturition	20(50)	5(12.5)**
5.	Dysuria	8(20)	2(5)*
6.	Retention	1(2.5)	0(0)*
7.	Hematuria	3(7.5)	0(0)*

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in all symptoms. So there is significant reducing of symptoms among the patients for the treatment of Kalladaippu. Hence it is concluded that the treatment was effective and **significant.**

Treatment for Kalladaippu:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

DISTRIBUTION OF CALCULI BASED ON LOCATION

S. No	Side	Before Treatment	After Treatment
		n%	n%
1.	Rt – Kidney	13(32.5)	10(25)*
2.	Lt – Kidney	5(12.5)	1(2.5)*
3.	Bilateral	20(50)	14(35)*
4.	Ureteral	2(2)	1(2.5) *

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in all sides. So there is a significant changes in distribution of calculi based on location. Hence it is concluded that the treatment was effective and **significant.**

ANNEXURE – VII

CONSENT FORM

ANNEXURE - VII

GOVERNMENT SIDDHA MEDICAL COLLEGE

ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE

CHENNAI – 600 106

CLINICAL STUDY ON **"VEDIYUPPU CHENDOORAM"** IN THE TREATMENT OF

"KALLADAIPPU" (UROLITHIASIS)

INFORMED CONSENT FORM

"I have read the foregoing information. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.

I consent voluntarily to participate in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care".

"I have received a copy of the information sheet/consent form".

Date:

Station:

Signature of participant:

Signature of the Guide: Signature of the Investigator:

அரசு சித்த மருத்துவக் கல்லூரி, சென்னை-106

அறிஞர் அண்ணா மருத்துவமனை, சென்னை

¸øÄ¨¼ôÒ நோய்க்கான சித்த மருந்தின் **(வெடியுப்பு செந்தூரம்)**

பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

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ANNEXURE – VIII

CASE SHEET PROFORMA

ANNEXURE - VIII

CASE SHEET PROFORMA FOR KALLADAIPPU

GOVT.SIDDHA MEDICAL COLLEGE&HOSPITAL, CHENNAI-106

POST GRADUATE DEPARTMENT BRANCH –I MARUTHUVAM

Duration: 2014-2016

OP No / IP No	:	Occupation	:
Ward No	:	Income	:
Bed No	:	Nationality	:
Name	:	Religion	:
Age	:	D.O.A	:
Sex	:	D.O.D	:
Address	:	Diagnosis	:

1. Complaints and duration :

2. History of present illness :

3. History of past illness :

4. Personal history :

5. Occupational history :

6. Menstrual history :

7. Personal Habits : Veg/non veg/smoker/Alcoholic/Tobacco chewer

8. Family History :

GENERAL EXAMINATION

Patient consciousness :

Body Built :

Nourishment :

Anemia :

Jaundice :

Cyanosis :

Clubbing :

JVP :

Tracheal deviation :

Pedal oedema :

Lymph adenopathy :

VITAL SIGNS

Body Temp :

Pulse :

Respiratory rate :

Blood Pressure :

Weight :

SIDDHA ASPECT

NILAM

Kurinji :

Mullai :

Marutham :

Neithal :

Palai :

PARUVA KALAM

Kaar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Muduenil :

YAAKKAI (Udal)

Vatham :

Pitham :

Kabham :

Kalappu :

GUNAM

Saththuvam :

Rajotham :

Thamasam :

PORI/PULANGAL

(SENSORY ORGANS)

Mei –Sensation :

Vaai – Taste :

Kan – Vision :

Mooku - Smell :

Sevi – Hearing :

.

KANMENTHRIYAM/KANNMA VIDAYAM [MOTOR ORGANS]

Kai- Dhaanam :

Kaal-Kamanam :

Vaai-Vasanam :

Eruvaai- Visarkkam :

Karuvaai-Aanantham :

UTHKAAYA ATHAKAAYAM

Puyam[forearm] :

Sayam[arm] :

Kaal[leg] :

Paaatham[feet] :

UYIR THATHUKKAL

A.VATHAM

Piranan :

Abanan :

Viyanan :

Udanan :

Samanan :

Nagan :

Koorman :

Kirukaran :

Devathathan :

Thananjeyan :

B.PITHAM

Anar pitham :

Ranjaga pitham :

Saathaga pitham :

Pirrasaga pitham :

Alosaga pitham :

C.KAPAM

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

UDALTHAATHUKKAL

Saaram :

Senner :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam/Suronitham :

ENVAGAI THERVUGAL

1.Naa :

2.Niram :

3.Mozhi :

4.Vizhi :

5.Sparisam :

6.Malam :

7.Moothiram

a)Neer Kuri :

b)Nei Kuri :

8.Naadi :

MALAM

Niram :

Edai :

Erugal :

Elagal :

MOOTHIRAM

1.Neerkuri

Niram :

Manam :

Edai :

Nurai :

Enjal :

2.Neikuri

MODERN ASPECT

Sytemic Examination

Inspection :

Palpation :

Renal angle :

Tenderness : Present/Absent

Radiation :

Percussion :

Auscultation :

Others Systems

Cardio Vascular System :

Respiratory system :

Central nervous system :

CLINICAL SIGN AND SYMPTOMS OF KALLADAIPPU

S.No	Symptoms	Before Treatment	After Treatment			
			10th day	20th day	30th day	40th day
1	Pain ✓ Site ✓ Radiation ✓ Character					
2	Nausea					
3	Vomiting					
4	Burning Micturition					
5	Dysuria					
6	Oliguria					
7	Haematuria					
8	Retention					
9	Fever					
10	Frequency of Micturition					

INVESTIGATION

1. BLOOD

TC, DC, ESR
Bleeding time, Clotting time
Blood sugar
Blood urea
Serum cholesterol
Serum calcium
VDRL

2. URINE

Colour
Turbidity
Albumin
Sugar
Deposits

- Epithelial cells
- RBC's
- Pus cells

Casts
Specific gravity
Urine culture and sensitivity

3. USG Abdomen and Pelvis

4. X-Ray KUB

TRIAL DRUG: veddiyuppu chendooram

Dose: 130- 260 mg, bd.

Adjuvant : mullangi kizhangu chaaru.

Duration of Treatment: 48 days

Pathiam (Do's and Don'ts)

Prognosis

Medical Officer Signature: **HOD**

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